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VOLUME XXXV OKLAHOMA CITY, OKLAHOMA, MARCH, 1942 NUMBER 3

Practical Refraction*

JAMES P. LUTON, M.D.
OKLAHOMA CITY, OKLAHOMA

My purpose in presenting this paper is to set forth briefly the steps which I consider necessary in the routine refraction as related to every day practice. In outlining these procedures I am not laboring under the delusion that they are the ones which will give the best results in the hands of every practitioner because it has been proven many many times that equally good refractionists may arrive at the same conclusion by methods which are not the same. With full realization of this fact I shall enumerate my own methods and at the same time try to cite some of the reasons which justify their use.

The first step, and one which is not open to controversy is a good history, not only of the present complaints, but of the past history and family history as well. The history alone, when carefully taken and properly analyzed will often not only rule in or out the eyes as the offending organs, but it will also present some general idea in regard to the type of refractive error present. The history should include information concerning the general health, and particularly anything concerning the infections of the upper respiratory passages and the para-nasal sinuses. I'm sure we have all seen patients with symptoms apparently due to refractive errors present, but who were not relieved by correction of that error. Then with more careful history taking and possibly with the aid of the rhinologist we find pathology in the nose or sinuses which proves to be the real cause of the symptoms. In many of these cases time, money, and perhaps embarrassment might have been saved by more attention to the history. The same may be true, though less frequently, of certain gastro-intestinal diseases and of certain types of pelvic pathology, not to mention numerous other possibilities. In my own experience this laxity in history taking is one of the most frequent stumbling blocks, simply because it is too easy to listen to the patient say, "My eyes give me a headache." and to allow it to go at that.

Before beginning the second step we check the manifest visual acuity both with and without correction if the latter is worn. This is followed by inspection with more or less oblique illumination from an ordinary 60 watt electric light bulb equipped with a reflector, and attached to an arm so that it may be moved to any desired position. Although it would probably be advised I do not use the loupe or condensing lens unless there is some suspicion of pathology in the cornea or anterior chamber. In case of doubt or if any pathology is visible to the naked eye I use an ordinary binocular loupe and a small hand slit lamp of the Shahan type to complete the inspection. At the conclusion of inspection it is the rule to observe the movements of the eyes in the six cardinal directions, for gross abnormalities in actions of the extrensic muscles.

From inspection we progress to the third step, that of determination of the near point of convergence and the near point of accommodation of each eye. The former is determined by having the patient fixate a small object, usually the point of a pencil, and while he is fixing the object it is gradually brought nearer to the eyes. The patient is asked to state when the object becomes double. During this procedure the eyes are carefully watched and the point at which one eye lags in convergence or gives up fixation entirely is taken as the near point of convergence, or NPC. The patient's eyes must be watched because it will frequently be found that he suppresses with one eye the minute it ceases to fix and he does not see double. The NPC is recorded in millimeters. Other information gathered by this simple procedure is a rough estimate of the grade of fusion present, and an indication as to which is the dominant eye. The non-dominant eye

*Read before the Section on Eye, Ear, Nose and Throat, Annual Session, Oklahoma State Medical Association, May 20, 1941, in Oklahoma City.

will usually be the one which gives up fixation first. The near point of accommodation is taken with each eye separately. We use the smallest letters on the near vision test chart, have the patient fix one word, and with the other eye covered we bring the card up to the eye under examination until the letters begin to blur. This distance is recorded in millimeters as the patient's near point of accommodation or PP . For those patients who are presbyopic or sufficiently hyperopic to require it we interpose a plus sphere and test in similar manner. The strength of the sphere must be recorded along with the distance in millimeters. The two, sphere in diopters and distance in millimeters, can be changed to either one or the other and added, but in practice we have found it more simple to record the two in combination. Determination of the NPC and PP are not only valuable in the immediate examination but they also offer a quick method in subsequent examination for determining any changes which may have taken place in the patient's ability to accommodate, thereby indicating the probabilities of the patient requiring a change in lenses. The PP also serves as a check on anomalies of accommodation and may put the examiner on his guard in looking for some pathological condition which would not otherwise be apparent. The method described here for determining the PP is used because it is simple, quickly performed, and gives the information desired in the ordinary routine refraction. We do not feel that it is necessary in the average case to go into the finer points of determination of the PP with the eye corrected for refractive errors because the information gained here is sufficient in most cases and we have it in mind to assist us in making deductions during the subsequent steps of the examination. If for any reason it is found necessary to make a more accurate determination this can be done after the refraction has been completed.

Our fourth step is measurement of the corneal astigmatism with the ophthalmometer. I realize that use of this instrument is considered superfluous by many men because it does not take into consideration the astigmatism which may be present in the lens. It does have the very definite advantage of measuring corneal astigmatism both in strength and as to axis very accurately in a majority of cases, and this is a very valuable adjunct to other findings if given proper consideration. It allows us to determine whether or not lenticular astigmatism is present and if so, how much is present and whether or not it coincides with the corneal astigmatism or tends to neutralize the same. If these factors are all considered properly in the final determination of the total error we feel certain that the prescribed glasses are much more likely to be comfortable and to serve the purpose for which they were intended. As a word of caution, there are three points at which the examiner is likely to be misled in use of this instrument if he is not constantly on his guard. I recall having allowed such errors to creep into my personal experience on more than one occasion. The first is that of placing too much confidence in the ophthalmometer reading and not searching carefully enough for astigmatism when the reading is negative. Relatively large errors may be overlooked if the examiner is lulled into a sense of false security in this way, and proper care is not exercised, both in the manifest and in the retinoscopic examinations. On the other hand it frequently occurs that the corneal astigmatism as recorded by the instrument is neutralized at least partially by accompanying lenticular astigmatism, and the patient may be over corrected. The third point of caution is the ability of some patients by the simple process of squeezing the lids to change the shape of the bulb, thereby changing the corneal curvature. I have seen this happen on numerous occasions while the measurement was being made, and the amount of such change may be very great. This of course may be prevented by asking the patient not to squint, and at the same time observing to see that he complys with the request.

Our fifth step is retinoscopic examination. I'm certain that we all agree that it is an indispensable part of every refraction, and particularly so in children who cannot tell us what they see. Even so, there is a wide variation of opinion as to the type of instrument used and the procedure to be followed. Due perhaps to my early training and habits developed at that time I have continued to use the plane mirror retinoscope electrically lighted from the handle. It is simple and convenient and seems to serve the purpose very well with the possible exception of inadequacy in the accurate determination of the axis of astigmatism. Users of the streak retinoscope seem unanimous in agreement that it is better for such determination, but until I have had more experience in its use the question will be left open in my mind. Our retinoscopic examinations are made with the refractor properly placed before the patient, the patient facing an object at 20 feet, in a semi-dark room. The working distance is about one meter. Examination is made with both eyes uncovered and at the start no lenses are present. From this point we determine whether the light reflex has a with motion in all meridians, if not, sufficient minus sphere is added to make the movements of the reflex with. Then plus sphere is gradually added until the first meridian shows a reversal. This is recorded and more

plus is added until the reversal becomes complete in all meridians, and the amount of sphere used at this point is recorded. The difference between these two points is the strength of the astigmatism and the meridian of the first reversal is the axis. When the refractor contains minus cylinders as is the case with our instrument, the opposite meridian is of course used as the axis and a minus cylinder of the strength indicated above is placed before the eye in combination with the total sphere used in obtaining the last reversal. From this we deduct plus 1.25 diopters of sphere from the total sphere used in bringing about reversal in all meridians. In other words we do not simply try to neutralize the movements, but rather try to bring about a complete reversal in order to be sure that we have gone as far as we should. This accounts for the extra plus .25 diopter deducted from the total.

We are now ready for the sixth step, namely, manifest refraction, which is of course the real proving ground of every examination. The preceding steps have given us a close approximation of errors of refraction present and an incite into the condition of the patient's eyes together with some important information in regard to the ability of his eyes to overcome the amount of error present. In short it gives us grounds on which to base our judgment of the accuracy of the subjective findings. We have gone a step further, in that we have prepared the patient for examination by fogging him to a considerable degree before the manifest examination has been started. This alone may uncover many cases of latent hyperopia.

Beginning with the retinoscopic finding placed before the patient and with one eye covered we procede in the usual manner to determine his manifest acceptance, first by attempting to add more plus sphere. If that is not accepted we try reducing the plus sphere or add more minus sphere as the case may be. As soon as the strength of sphere has been satisfactorily determined we turn to the cylinder, first testing the various strengths above and below that in the instrument, then by changing the axis to various points from five to 20 degrees in each direction from the original. At this point we resort to the use of a procedure which I feel is most valuable in the accurate determination of the strength and axis of the cylinder, that is the Jackson cross cylinder. A complete description of the method is not possible here because it is a subject which would take too much time. Since returning to its use some two or three years ago after throwing it in the discard for a year I have become so dependent upon the results that I would not like to be required to do a manifest examination without it. It has solved several difficult problems for me which had remained virtually unsolved during that period of time in which the cross cylinder went unused. It is very valuable in getting away from overcorrection with minus cylinders and at the same time it is the most accurate and positive manifest method of determining the axis I have yet found. Following use of the cross cylinder we again check the sphere then re-check the patient's astigmatism on the radial chart, more perhaps through force of habit than for any real reason, because I cannot remember making any major change based on results gained from use of this chart. In all fairness to the method it may be said that the chart does occasionally cause one to return to the cross cylinder and other manifest examination in order to re-check the results. As a final check on the sphere chiefly in order to prevent fogging the patient, we always use the bichrome test. It has the advantage of being a very sensitive test, quickly and easily performed.

Following the manifest examination we determine the state of muscle balance with the Maddox rod and red glass, then the ductions by prisms and the muscle light at 20 feet. This phase of the examination will not be discussed here since it was taken up in some detail on a paper read before this group at the meeting last year. Suffice it to say that we consider it a routine procedure to be used in every case of refraction when two useful eyes are present.

In the eighth step we check by manifest examination, the near vision using the age of the patient, the amount of accommodation present as determined at the beginning of the examination, and our judgment based on past experiences as guides to the procedure. Some men have been able to use successfully dynamic retinoscopy as an aid here, but the reflex obtained has been too poor and too rapid in motion to be followed in practical work. There are only two conditions under which the manifest findings at near greatly influence our later decisions. The first is in presbyopes or in those patients who have marked weakness of accommodation from some other cause. The second is a reduction of cylinder strength in those cases of lenticular astigmatism which are reduced by the act of accommodation.

Following the above procedures we make an examination of the internal eye with the ophthalmoscope, then we are ready to decide whether the patient should be examined under cycloplegic or simply be re-checked on a later date. In general, all patients under 15 years of age are examined under atropine, those above 15 and up to 30 years are examined under homatropine, and those older are re-checked the following day. In the

absence of glaucomatous states, paresis of accommodation and etc. we do not hesitate to use homatropine on older patients, even up to 45 or 50 years if conditions seem to indicate its use. For the most part a repetition of the retinoscopic and the manifest examinations is sufficient and this we always insist upon using, even though it may mean an out of town patient makes an extra trip. This second examination is made, if possible, early in the morning or at such time as the eyes have had plenty of rest, and the patient is not tired and nervous. If the findings at this examination do not agree reasonably well with those of the first examination we may resort to a cycloplegic or to a third examination, depending upon the conditions involved. If the findings do coincide, as is usually the case, we feel reasonably secure in our judgment as to the final prescription.

Pernicious Anemia

WANN LANGSTON, M.D.

OKLAHOMA CITY, OKLAHOMA

In the preface to his paper "On the Constitutional and Local Effects of Disease of the Suprenal Capsules" before the South London Medical Society in 1849, Thomas Addison said, "For a long period I had from time to time met with a very remarkable anaemia, occurring without any discoverable cause whatever—cases in which there had been no previous blood loss, no exhausting diarrhea, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease." This he designated as "Idiopathic Anaemia." To quote further: "The disease presented in every instance the same general character, pursued a similar course, and with scarcely a single exception, was followed, after a variable period, by the same fatal result." "It occurs in both sexes, generally, but not exclusively, beyond the middle period of life, and, so far as I, at present, know, chiefly in persons of somewhat large and bulky frame, and with a strongly marked tendency to the formation of fat. It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to his earliest feeling of languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted; the pulse, perhaps, large, but remarkably soft and compressible. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness and breathlessness on attempting it; the heart is readily made to palpitate; the whole surface of the body present a blanched smooth and waxy appearance; the lips, gums and tongue seem bloodless; the flabbiness of the solids increases; the appetite fails; extreme languor and faintness supervene, breathlessness and palpitation being produced by the most trifling exertion or emotion; some slight edema is probably perceived about the ankles; the debility becomes extreme, the patient can no longer rise from his bed, the mind occasionally wanders, he falls into a prostrate and half torpid state, and at length expires."

Addison goes on to say that upon postmortem examination he failed to find "any organic lesion that could properly or reasonably be assigned as an adequate cause of such serious consequences." He suspected some form of fatty degeneration, and in one case observed such a change in the heart, the semi-lunar ganglion and the solar plexus.

This classical description of the disease was not the first reference to the condition. J. S. Combe had in 1822 described what seems to have been a case, in which he referred to remissions of the symptoms.

In 1851 Barclay published a case in which he, for the first time, recorded soreness of the tongue. In 1870 Fenwick described atrophic changes in the mucosa of the stomach and intestines, and he related the blood changes to this atrophy. In 1871, Biermer apparently unfamiliar with Addison's report of 22 years before described a "new" disease which he named "Progressive Pernicious Anemia." Biermer, like Addison, stressed the point that the disease occurred without discoverable cause, but pointed out that it was especially prevalent among the poorer classes, an observation which William Hunter did not confirm. Hunter was equally in error when he made the statement that the condition is a "chronic infective disease." Hunter did, however, make an important contribution in stressing the atrophy of the gastro-intestinal mucosa, and in emphasizing the importance of Glossitis and Stomatitis. He went to considerable length in describing the glossitis even to the periodicity. We still speak of "Hunterian Glossitis" in connection with the diagnosis of Pernicious Anemia.

In 1874-75 Immerman made an important

contribution in separating a primary pernicious anemia for which no cause could be found as described by Addison and by Biermer, and a secondary, associated with other disease states. The latter group gradually grew to include the anemias associated with pregnancy, carcinoma of the stomach, parasitic infestation and dietary deficiencies.

In 1889 Leichtenstern described disseminated foci of degeneration in the posterior and lateral columns of the spinal cord in cases of severe anemias and designated the condition as "Anaemic Spinal Disease." Five years before he had reported three cases of pernicious anemia with tabes dorsalis.

In the last quarter of the last century the blood picture began to excite interest; macrocytosis and poikilocytosis were observed, and American workers noted hyperactivity of the bone marrow and the presence of nucleated red cells in the circulating blood. During the same period absence of hydrochloric acid was discovered in cases of pernicious anemia.

During the first quarter of the twentieth century no important contribution to our knowledge of pernicious anemia was made. And then, in 1926, came the announcement of the epoch making discovery of Murphy of the effect of liver feeding upon cases of pernicious anemia. The effect of this discovery was to stimulate investigation as nothing before had; a tremendous literature appeared; the entire field of hematology was intensively studied and a more complete understanding of blood dyscrasias was acquired than in all the previous years.

Thus, during the period of a hundred years, from Combe in 1822 to Murphy in 1926, gradually was constructed the picture of pernicious anemia as we know it today, namely, a progressive anemia with the constitutional disturbances associated with blood impoverishment, atrophy of the gastrointestinal mucosa, with achlorhydria; Hunterian Glossitis, and Stomatitis; the presence of macrocytosis; the presence of "Anaemic Spinal Disease," subacute combined degeneration of the spinal cord; and the characteristic response of the disease to specific therapy. A pernicious progressive fatal disease has been transformed into a benign condition, the most satisfactory to treat of all chronic incurable pathological processes.

RECOGNITION

The diagnosis of Pernicious Anemia depends mainly upon the recognition of abnormalities in three systems. The finding of such in one system leads to suspicion; in two, to diagnosis; in all three, to certainty. Finally, response to therapy removes all doubt if any exists.

First, the Digestive System: Anorexia, indigestion, nausea and vomiting; intermittent diarrhea; atrophy of the papillae of the tongue, glossitis and stomatitis; and the absence of hydrochloric acid in the gastric secretion. In former times the appearance of the tongue played a much greater part in diagnosis than now. In no other condition perhaps, is the appearance of the tongue so characteristic as in pernicious anemia; and in no condition, so far as I know, does the patient complain more bitterly. Often this glossitis and stomatitis constitute the very first abnormality noted. The edges and frequently the entire surface of the tongue are inflamed and have a beefy red or raw appearance; later the tongue may take on a perfectly smooth character, the entire dorsum being denuded of papillae. The buccal cavity may have the same appearance. Hunter called attention to the periodicity of this involvement of tongue and mouth, periodicity being a characteristic of the disease itself. Achlorhydria is said to occur universally.

Second, the Blood: The Anemia is characteristically hyperchromic and macrocytic. The hemoglobin is high, relative to the number of red cells. If one studies the stained smear he will be struck with the appearance of cells that are abnormally large, some that are smaller than normal, and many that are abnormally shaped—poikilocytes. Occasionally nucleated red cells will be seen. If a vital stain preparation is made reticulocytes will be seen in excess of the normal number, one percent. The mean corpuscular volume will be increased. There likely will be a leucopenia, and the neutrophils will be seen to have multilobular nuclei. The icteric index is frequently increased. Following specific and adequate therapy the reaction is characteristic—a rapid increase in reticulocytes, and slower rise in red cells and hemoglobin, with a drop in reticulocytes as red cells and hemoglobin approach normal. If the bone marrow is examined it will be found to be hyperplastic, and megaloblasts will be seen to predominate the picture.

At this point one may inquire as to a possible etiological relationship between these two systems, the blood and the digestive. In these last 15 years the investigations of Whipple, Castle, Minot and Murphy, Sturgis and Isaacs, and others have thrown much light upon this subject. Our present information may be summarized as follows:

The red cells are formed intravascularly in the capillary bed of the bone marrow, mainly lodged in the flat spongy bones. As the immature cells pass along the assembly lines at least two substances are added. One of these is hemoglobin, and if iron is not available iron deficiency anemia results. The

other is a principle required for the proper maturing of the red cells. This has been designated the Erythrocyte Maturing Factor. If not available another type of deficiency anemia results, characterized by immaturity of the red cells. This is the anemia of Pernicious Anemia and the erythrocyte maturing factor has also been called the Anti Pernicious Anemia Factor or Principle. This principle normally is stored mainly in the liver and is supplied to the red cell factory on demand. If the storage be interfered with by extensive damage to the liver, an anemia resembling pernicious anemia may ensue. This Erythrocyte Maturing Principle is produced in the normal stomach by the interaction of two factors, one resident in the normal gastric mucosa and designated as Intrinsic Factor, and the other supplied in the food, principally protein, and called Extrinsic Factor. If the former is lacking due to atrophy or destruction of the gastric mucosa no Erythrocyte maturing factor is available; if there is deprivation of the necessary food elements there is also an insufficiency of the E. M. F. Such a deficiency may be corrected by the administration of adequate quantities of normal liver, in the form of food, as dried extracts administered orally, as more or less purified and concentrated extracts given parenterally, by the administration of normal gastric juice containing partially digested protein, or by the oral administration of preparations of normal gastric mucosa. Regardless of how it is supplied, E. M. F. is stored in the liver until required. The Intrinsic Factor seems to be lodged mainly in the mucosa near the pylorus. When this part of the mucosa is destroyed extracts of the liver fail to produce a reticulocyte response in pernicious anemia patients, while extracts from the livers of individuals in whom the entire gastric mucosa except near the pylorus is destroyed do produce such a response.

Thus there seems to be a relationship between the gastro-intestinal pathology in so far as it is the cause of deficiency in intrinsic factor, and the blood picture in Pernicious Anemia. Whether this same deficiency is responsible for the glossitis-stomatitis, I do not know. As will be pointed out later this seems a possibility; and the evidence seems to point to this gastro-intestinal pathology as the primary etiological factor.

The third system likely to be involved in Pernicious Anemia is the Neuro-muscular. Addison reported fatty degeneration of muscle and nerve plexuses. Leichtenstern recognized degeneration of the postero-lateral columns of the spinal cord in his "Anemic Spinal Disease." We now recognize signs of sub-acute combined degeneration of the spinal cord in nearly 50 percent of all cases of primary pernicious anemia, and in some cases of other blood dyscrasias and secondary macrocytic anemias.

Sub-acute combined degeneration of the spinal cord may be defined as a progressive degenerative process affecting mainly the posterior columns and the pyramidal tracts of the cord, and usually associated with pernicious anemia.

The manifestations of involvement of the posterior columns are, first, paresthesias, numbness, tingling, sensations of coldness, burning and pricking, sometimes girdle sensations and back pain and varying degrees of anesthesia; second, ataxia and incoordination due to loss of tendon, joint or muscle sense, unsteadiness of gait particularly at night, loss of sense of position of the smaller, distal members, as the feet and toes; astereognosis with manual clumsiness, etc.; third, loss of vibratory sense, especially in distal members particularly below the knees.

Positive plantar reflex and exaggerated tendon reflexes and clonus indicating pyramidal tract involvement occur in a considerable percentage of cases; loss of sphincter control occurs in some. As the disease advances spasticity and contractures sometimes supervene.

This neurological syndrome must be differentiated from multiple sclerosis, tabes dorsalis, cord tumor and peripheral neuritis. This should not be difficult when one considers that this syndrome is usually associated with macrocytic anemia, achlorhydria and glossitis and stomatitis, findings not usually present in the others. Pernicious anemia then should not be difficult of recognition, since macrocytosis is practically always present by the time either neurological symptoms or findings appear.

At this point one may inquire as to the relationship of the nervous system lesion and the pathological processes in the other systems involved. The answer is not yet. It seems probable however that impairment in the function of producing Intrinsic Factor in the gastric mucosa precedes the other manifestations. In many cases glossitis and stomatitis are the first abnormal findings of which the patient complains, but upon investigation, achlorhydria and anemia with macrocytosis already are present. In other, and a large percentage of cases, the symptoms of anemia will be the first complained of, but again achlorhydria will be found. Again, in not a few cases neurological symptoms and signs will be the first obvious manifestations, but still the ever present macrocytic anemia and the achylia. Then this question arises: Just as the peculiar type of anemia must be due to the absence of anti-pernicious anemia principle, due in turn to absence of Intrinsic

Factor (in most cases), does it not seem likely that the glossitis and stomatitis on the one hand, the spinal cord degeneration on the other, may also be due to this deficiency?

MANAGEMENT

The modern streamlined treatment of the average case of pernicious anemia consists solely of the parenteral administration of adequate amounts of potent liver extracts to correct the deficiency—to maintain a blood count at normal level or a little above. This can be done by administering liver extract containing two units per cubic centimeter intramuscularly, six to ten units daily for a few days, then at gradually lengthening intervals, until finally only a dose a week or one in two weeks may be needed. More highly purified and concentrated extracts are now available, but one feels that perhaps they are not as effective as the cruder preparations. Iron is not required, and mixtures of iron and liver orally constitute an expensive, inconvenient, disagreeable, and unrational method unless there is an associated iron deficiency which is unusual. Hydrochloric acid in most cases can be dispensed with. Vitamin B. is of questionable but probable advantage. Naturally a patient on this regime must be given a well balanced dietary such as a normal individual would receive. In other words, one is dealing with a deficiency disease, and the one problem is to correct the deficiency by adequate replacement therapy. Adequate rest is important.

RESULTS OF MANAGEMENT

What may one expect to happen to the various symptoms and manifestations on such a regime? The answer to this question may throw some light upon the relationship of system abnormalities.

One of the first as well as one of the most striking effects of adequate therapy is the evidence of a return to normal function of the bone marrow. This is manifested by the remarkable increase in the number of reticulocytes in the blood, sometimes a rise of from one percent to 12 percent or 15 percent within a week, and a slower but notable increase in the hemoglobin readings and in the red cell count. As the last two approach normal, the reticulocyte count also approaches normal, that is it rapidly falls to a normal one or two percent. At the same time there will be noted a reduction in the number of macrocytes, poikilocytes and microcytes—the entire blood picture approaches normal. If therapy be discontinued the blood picture will again become abnormal.

This seems to establish definitely the fact that the liver extract contains a substance needed for the proper maturing of the red blood cells in the bone marrow. It has been reported that extracts from the liver of an adequately treated case of pernicious anemia, will produce a reticulocyte response in cases of pernicious anemia, while extracts from livers of untreated cases will not produce such a response, thereby increasing the evidence that E. M. F. is stored in the liver.

Equally as prompt as the improvement in the blood picture is that of the sore tongue and mouth when present. One has seen patients who were complaining bitterly of pain in the tongue greatly relieved after one or two large doses of liver extract intramucsularly. One of the most gratifying results of this form of therapy is the prompt relief of this, one of the most annoying symptoms of the disease. Not infrequently the mouth and tongue are completely relieved within ten days from the first dose of liver. But if therapy be discontinued the relapse is prompt. Again, a direct etiological relationship is suggested between the deficiency in Erythrocyte maturing Factor, or at least, between Intrinsic Factor deficiency and the pathology of tongue and mouth.

The results of therapy have not been nearly so striking or satisfactory in the cases of pernicious anemia with subacute combined degeneration of the cord. Much larger dosage is required, and a much longer period of time elapses before the benefits are obvious. There is also a great variation in the responses of various cases; as would be expected, those with milder symptoms, of shorter duration respond much more readily than the more severe cases and those of longer duration. But persistence in treatment with large doses of liver will greatly benefit a very large percentage of even those with high grade disability. One can give a favorable prognosis in the severe cases of short duration, but must be conservative about those of several years' standing.

Practically every case of pernicious anemia is troubled with paresthesias. Some of these undoubtedly are associated with sub-

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acute combined degeneration; others probably are not on this basis. The latter usually disappear promptly with adequate liver therapy; the former are likely to persist for a long time and may not disappear at all.

Loss of vibratory sense is one of the most constant of neurological findings in pernicious anemia. With adequate therapy at least a partial recovery can be expected in many cases, even with severe degenerative lesions. The same is true of sense of position, astereognosis and superficial sensation. A large percentage of patients with disturbances of gait show marked improvement, some are completely relieved. In those who remain handicapped re-education of muscles has been a most satisfying adjunct to liver therapy.

In like manner persistent adequate therapy has had a very satisfactory effect upon the lateral column symptoms in that deep and superficial reflexes have been at least partially restored to normal, and spasticity relieved. Even those with bladder and bowel involvement have been greatly benefited, in some, normal function has been restored.

It has been suggested, and it has been my observation that in cases with neurological symptoms refractory to liver therapy improvement has been enhanced by the administration of preparations of gastric mucosa, such as ventriculin. Here, I believe, is the chief indication for any mixed therapy in pernicious anemia.

SUMMARY

1. The history of the development of our knowledge and present conceptions of Pernicious Anemia has been sketched briefly.
2. Symptoms and findings associated with Digestive, Blood and Neurological systems have been enumerated.
3. The relationship of disturbances in these systems to each other has been suggested.
4. Adequate management has been very briefly outlined and results that may be expected have been mentioned.

The Present Status of Vitamins in Neurology and Psychiatry

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The vast, increasing literature on vitamins, the high pressure advertising of pharmaceutical firms, and the public curiosity and "vitamin consciousness," which has been aroused by the press and radio, have all combined to make the physician more interested in vitamins. But the busy physician who attempts to bring himself up to date on this subject finds himself confronted with thousands of articles; with a confusing terminology; and an array of claims and counter-claims by different workers. At least the writer had that experience and decided therefore to try and pick out the new, essential and verified facts about vitamins, especially those facts which relate to the fields of Neurology and Psychiatry. Much of our present knowledge was learned from animal experiments and later was confirmed by similar clinical observations on humans. But the results in animals do not always coincide with those in man. A number of vitamins, for example, whose deficiency in animals causes certain deficiency syndromes, have as yet no clinical application in humans. Many complexities and unknowns face us. For instance, certain work shows that heredity plays a certain role. Further the *exact* daily reqirement of any vitamin is still unknown. This is understandable when we consider the influences of absorption, storage, and destruction, not to mention the marked variability in the minimal and optimal vitamin requirements for any particular individual. Also our problem is further complicated because single vitamin deficiencies seldom exist. Apparently too, certain safety factors operate to prevent the development of a deficiency state: (1) there is quite a margin of safety between the beginning of a deficiency disease and death; (2) there is a certain amount of storage of vitamins, especially of A and D, and (3) the body excretes less vitamin when a deficiency in that vitamin exists. In America gross deficiency states are uncommon and usually recognized. But the subclinical and latent deficiency states are very common and usually unrecognized by the physician. Four sets of factors operate to produce deficiency states: (1) an inadequate intake of the vitamin; (2) an increased need for that vitamin; (3) diminished absorption or utilization of that vitamin by the body; and (4) increased destruction or excretion of the vitamin. We shall be interested in the nature, sources, requirement, action and function, clinical features, and therapy in the case of each vitamin. For

the sake of brevity the word "vitamin" will be left out as much as possible and designations such as "A," etc., will be used instead.

VITAMIN A

A is related to *carotene*, the latter being a yellow substance in certain plants. That is, A begins as carotene; the carotene is converted by a liver enzyme into A, and the liver is a storage depot for carotene. A is found abundantly in fish liver oils, egg yolks, butter and cream. A is a catalyst, which is essential in maintaining the normal state of epithelial tissues. It is in the other words a so-called epithelial protective vitamin, and is necessary for the continued health of all tissues of epiblastic origin. Thus, the brain and central nervous system, the retina, the skin and its glands and mucous membranes require A for their normal function. Where A is deficient the epithelial tissues show thickening or keratinization, followed by secondary infection and destruction. A is also a precursor of visual purple, which is the photosensitive substance in the dark retina. Deficiency of A thus causes difficulty in dark adaptation or night blindness, which can be determined quantitatively by the ophthalmologist with his biophotometer. Apparently 2,000 to 6,000 units of A represent the daily requirement.

The grosser manifestations of A deficiency are often picked up by the physician. But recent studies of large groups have demonstrated that many cases of mild to moderate A deficiency go unrecognized and that the individual himself may be unaware of its manifestations. Many people because they work under excessive light, eg., actresses, beauty operators, etc., develop early signs of A deficiency.

OCULAR SYMPTOMS—The A deficiency state begins with burning, itching, and dryness of eyes; photophobia; difficulty in reading over 10 to 15 minutes, especially at night; momentary attacks of blurring of vision at end of day; difficulty when viewing movies; dizziness; or even complete night blindness. Difficulty in driving at night may be responsible for auto accidents. The medico-legal aspects of this condition have not been realized. No such test is yet required of auto drivers or of aviators. The A deficiency state may progress to granulation and redness of the conjunctiva, softening of the cornea and blindness. In other types the skin especially over the extensor surfaces of the forearms, legs and thighs, shows keratinization of a hard, papular character that resembles goose-skin. There is some evidence that keratinization of the epithelium of the renal pelvis leading to urinary calculi may occur. Deficiency "A" also may lead to dental caries. As therapy, A or carotene in oil or oleum percomorphum is used; giving 10,000 to 50,000 units daily.

B COMPLEX

This complex contains at least 12 fractions, four of which are now available for clinical use, namely: (1) B_1 or thiamin chloride; (2) B_2, G, or Riboflavin; (3) Nicotinic acid or the P.-P., factor and (4) Pantothenic acid. Two other less well understoood B fractions are (5) Pyrodoxin and (6) Yeast Adenylic Acid.

(1) B_1 OR THIAMIN CHLORIDE

B_1 is found in yeast, milk, egg yolk, pork, etc. The daily requirement is about one to two mg. The kidney, liver and heart have a low storage capacity for B_1. B is a catalyst essential in the metabolism of carbohydrates, thus playing a part in the oxidation of lactic and pyruvic acids. Thus in B_1 deficiency we have abnormalities in pyruvic acid metabolism which is partly responsible for the disturbances of the central nervous system, and for the degeneration of the peripheral nerves. Clinically the gross B_1 deficiency states such as Beriberi with its symmetrical peripheral neuropathy and characteristic cardiovascular disturbances are familiar to the physician. Whereas, the milder and partial deficiency states often escape recognition until a severe state has blossomed out. Therefore, it behooves the physician to be on the lookout for these milder states. The milder states begin either with neurological manifestations or with a neurasthenic-like picture. Jolliffe classifies the neurological manifestations into the (1) suggestive, (2) mild, (3) moderate and (4) severe stages. In the suggestive stage the patient complains of anorexia, fatigue, heaviness of the legs, calf muscle cramps, and paresthesias in the toes and fingers. Objectively we find: calf muscle tenderness, plantar hyperesthesia and loss of vibratory sensation in the toes. The mild stage is characterized by absent ankle jerks. The moderate stage is marked by an advance in the sensory and motor changes; loss of the knee jerks; impaired position sense in the toes; atrophy of calf muscles and foot-drops. In the severe stage, we find involvement of the upper extremities; of the spinal cord; or of the cranial nerves. Certain sub-clinical B_1 deficiency states may simulate a neurotic or psychotic picture and lead to a mistaken diagnosis of a neurosis or psychosis. Here occur such symptoms as: a loss of weight and strength, indigestion and dyspepsia, epigastric burning, constipation, diarrhea, abdominal cramps, irritability, apprehension, depression, crying spells, flight of ideas, mental confusion, etc. The simulated neurotic and psychotic pictures are characteristic of thiamin, riboflavin, or nicotinic acid de-

ficiency states, or of a combination of all three of them. Therefore, it is wise to give all three vitamins in such cases. As therapy in the B_1 deficiency states children receive five mg. thiamin intravenously or intramuscularly daily, plus ten mg. by mouth. For adults 20 to 100 mg. doses may be necessary. Such conditions as tinnitus, tabetic pains, the cerebral complications following recovery from infectious diseases, and the withdrawal symptoms that occur in the treatment of alcoholic and morphine addicts have all apparently been benefited by B_1 therapy. But large doses may cause herpes zoster. If any intense burning pain develops in any part of the body during thiamin therapy, then B_1 must be discontinued immediately. Apparently, suprarenal cortex given in conjunction enhances the effects of B_1.

(2) B_2, G, OR RIBOFLAVIN

The requirement is probably about two to five mg. daily. B_2 is essential in the oxidation processes of the cells. B_2 deficiency usually begins with "Cheilosis," ie. a pallor of mucosa of lips in the angles of the mouth, followed by superficial transverse fissures and reddened, macerated areas in the angles of the mouth; and a greasy desquamation in the nasolabial folds, in the alae nasi, in the vestibule of the nose, and on the nose. Ocular symptoms where they occur include: itching and burning of the eyes, photophobia, excessive dryness of the conjunctiva or excessive lacrimation, keratitis, vascularization and opacities of the cornea, and abnormal pigmentation of the iris, failing vision, etc. These ocular symptoms wax and wane and are worse in summer and winter. As therapy three to five mg. Riboflavin orally or intravenously in four to six doses; plus 50 to 75 gm. of brewer's yeast is sufficient.

(3) NICOTINIC ACID

A gross deficiency of nicotinic acid seemingly leads to Pellagra with its well known oral lesions, gastro-intestinal disturbances and mental aberrations. Not so well known, however, is the fact that each of these three characteristic symptom complexes, may occur alone or in any possible combination and therefore, the basic condition may easily be overlooked by the physician. When mental aberrations occur singly, many such patients are labeled neurotic, etc. Recently an "encephalopathic syndrome" due to nicotinic acid deficiency has been described. This syndrome shows the following symptoms: (1) clouding of consciousness, (2) cog-wheel rigidities of the extremities, (3) uncontrollable grasping and sucking reflexes; (4) cerebellar symptoms and (5) catatonia. Therapeutically 500 to 1,000 mg. of nicotinic acid daily or 50 to 80 mg. of it intravenously daily in 10 to 15 mg. doses, dissolved in sterile physiologic saline solution is indicated. Also apparently the addition of nicotinic acid or of the amide to the diet produces a much larger food intake and better weight for any individual.

(4) PANTOTHENIC ACID

Is little understood yet.

(5) B_6 OR PYRODOXIN

Is still in the experimental stage. There is some evidence that it benefits cases of muscular dystrophy and lessens the muscular rigidity and weakness in cases of paralysis agitans. It is also claimed that extreme nervousness, insomnia, irritability and stomach cramps respond to it. As therapy 50 mg. of pyrodoxin in sterile physiologic saline solution is given daily.

(6) YEAST ADENYLIC ACID

This is another factor about which little is yet known. It is said to clear up intense burning of the oral mucous membrane with malnutrition, in a patient showing no evidence of pellagra. The dose is 200 mg. by mouth. Also this factor seems to increase the effectiveness of nicotinic acid in treating pellagra.

C OR ASCORBIC ACID

Occurs principally in milk, parsley, peppers, raw liver and watercress. C is a powerful reducing substance, essential in the formation and maintenance of the intercellular substance especially that of the blood vessels, dental forming organs and connective tissues. C also plays some indefinite role in respiratory function. At least 0.8 mg. of Ascorbic acid for each kilogram of body weight is used daily by healthy subjects. C seems to be stored in the adrenal cortex. The gross C deficiency state is Scurvy, with which the physician is familiar. The pathologic change here is in the intercellular substance. Especially in that of the blood vessels and bones, with softening which leads to hemorrhages into the subcutaneous, subperiosteal, gingival and other tissues. But subacute scurvy is generally unrecognized as the cause of a low state of health with fatigue, sallow complexion, and fleeting pains in the bones. Dental caries is thought to be one of the earliest signs of latent scurvy. C is essential also for the functional activities of certain cells necessary for the formation of the teeth. C deficiency also has some relationship to certain states of hemorrhagic diathesis and to certain states of macrocytic anemia.

VITAMIN D

D is found principally in liver oils of fish, egg yolks, butter, milk, and cream. D plays a part in the metabolism of calcium and phosphorus, having to do with the retention of calcium and phosphorus in the bones. Normal hepatic function seems to be neces-

sary to insure the proper metabolism of Vitamin D. Clinical D deficiency states show up principally in the form of rickets, tetany, dental caries, and possibly osteomalacia. In regard to therapy at present massive doses of D are given. Usually as much as 500,000 international units of D_2 (calciferol) are given weekly by mouth in an oily solution with milk. One-half the dose is given on the third day and the other one-half on the seventh day of each week. In giving massive doses of D there is some danger of causing hypervitaminosis D. Symptoms of this condition are as follows: First, there is cell injury, followed by excessive deposition of calcium, but the process is reversible and reparable if the administration of vitamin is discontinued immediately. Any evidence of renal impairment or of ateriosclerosis should be a definite contraindication against excessive doses of D. Symptoms of toxicity include anorexia, and gastro-intestinal and urinary irritability. One case recently died presumably of D hypervitaminosis.

VITAMIN E

E is found principally in wheat germ oil, in lettuce, spinach, and cabbage. Recently synthetic concentrates of the alpha and beta-tocopherol forms of E have been put on the market. A lack of E in the male is thought to cause destruction of the germinative cells. And its lack in the female is thought to be connected with spontaneous abortions and sterility. At any rate there appears to be some relationship between E and the ovarian hormone. Apparently anterior pituitary substance can be substituted for E. There is possibly a definite relationship between the inadequate intake of E and the normality of the cross-striated musculature of the body. In recent months for instance Wechsler, Stone and other workers reported remarkable cures and improvement in cases of amyotrophic lateral sclerosis, and in progressive muscular dystrophy by giving vitamin E. But other workers have gotten no results. The writer has had the opportunity of following a large series of both types of cases for the last nine months, and so far feels rather pessimistic as regards any beneficial results to be obtained by vitamin E in the above mentioned conditions. As therapy wheat germ oil is given in any amount from one dram t.i.d., upward. Ephynal or synthetic tocopherol is given daily in doses of 30 to 50 mg. either orally or intravenously. In addition foods rich in E like spinach, cabbage, lettuce etc., are given. And finally two 5 grain pills of bile salts are also given daily.

VITAMIN K

Is only being mentioned as its uses lie outside the fields of neurology and psychiatry.

Tularemia: A Report of Ten Cases*

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The importance of tularemia to the general practitioner is recognized by the ever increasing reports of cases occurring in the United States and many foregin countries. The recent observation of several cases prompted this report and I believe it would be well to review briefly some of the more pertinent aspects of the disease and the recent literature concerning it.

Historically, the disease was first described in 1910 by Dr. George McCoy of the Public Health Service in reporting a "plague like" outbreak among ground squirrels in Tulare county, California. McCoy and Chapin discovered and described the causative organisms in 1912, calling it Bacterium tularense after Tulare county where the disease was first observed. Dr. Edward Francis also of the Public Health Service, showed in 1919 that humans acquired the disease from infected animals and called it tularemia.

The Oklahoma State Health Department reports four cases in 1936, ten in 1937, 50 in 1938, 54 in 1939, 62 in 1940. The total number of deaths was 25 or approximately 14 percent. Apparently many of the milder cases were not reported. It should be noted that the number of wild rabbits has increased markedly in the past few years in this region. For the country as a whole, 2,200 cases were reported in 1939 with 140 deaths or approximately seven percent.

The commonest source of infection is skinning infected rabbits with bare hands, or handling their pelts, and this accounts for over 90 percent of human cases in the United States. Many other animals contract the disease and are potential sources of infection. Tick bites, of the wood tick principally, rank second as the infective source. Eating

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insufficiently cooked wild rabbit meat has been reported as a cause of numerous cases in the United States which resulted in an exceptionally high mortality. This appears to be the source of many cases falling in the typhoid class.

Francis recognized four clinical types of the disease and this classification is still in use today.

1. Ulcero-glandular. The primary lesion is a papule of the skin, commonly on the fingers, which later becomes an ulcer accompanied by enlargement of the regional lymph nodes. Visible lymphangitis is strikingly absent and when present is due to secondary infection. The majority of human infections are contracted as the result of the organisms entering through a skin wound, inflicted at the time of infection or shortly before or shortly after the germs get on the skin. Sometimes the infection penetrates the apparently unbroken skin of the hands, but usually there is a minute cut or scratch through which the germs enter the body.

2. Oculo-glandular. The primary lesion is a conjunctivitis with enlargement of the regional lymph nodes, chiefly the preauricular and retromaxillary glands. Presumably the infection is conveyed to the eyes by fingers soiled by rabbit blood.

3. Glandular. Regional lymphadenopathy alone occurs in this type, no primary lesion being demonstrable. It is probable that the primary lesion healed early in these cases or that it was so small that it made no impression on the patient. Patients should be questioned closely on this point as many of them do not present themselves until the second or third week of the disease, frequently long after the primary lesion has healed.

4. Typhoid type. In this type there is neither glandular enlargement nor primary ulceration. Septicemia dominates the picture, pneumonia is more frequent, and the mortality is highest. This form is common among laboratory workers who have performed necropsies on infected animals or handled infected ticks.

Most cases can be placed in one of these four groups, however the disease assumes such bizarre forms at times that accurate classification is difficult.

DIAGNOSIS

The diagnosis of tularemia is not always made easily or with certainty. This is particularly true of the typhoid type which may exist as a blood stream infection without regional lymphadenopathy or local lesions. Patients presenting atypical pneumonias, unexplained pleural effusions, unexplained toxic rashes or prolonged fever should be investigated with the diagonsis of tularemia in mind. The great majority of cases, however, fall into the ulcero-glandular group and the diagnosis should be easy if its presence is suspected. The laboratory is frequently called upon to make the diagnosis in obscure cases and to confirm a clinical diagnosis in obvious cases. The agglutination test for the detection of agglutinins in the blood is the most widely employed of the diagnostic tests and is highly reliable. It is useless, however, in the early stages of the infection, for agglutinins are never demonstrable during the first week and occasionally may not appear until the third week. Usually a titer of about 1:640 or above is reached by the third week and this persists for years afterwards. Foshay reports a case showing a strongly positive test 33 years after recovery. On the other hand, Moss and Weilbaecher have observed an absence of agglutinin titer in severe cases. They report six fatal cases in which the diagnosis was established only at necropsy.

Foshay has introduced the intradermal test reaction as an aid in early diagnosis. This is done by injecting intradermally .05 cc of a specially prepared B. tularense suspension. A positive reaction has the appearance of a positive tuberculin test and like the latter usually requires 48 hours for its development. The chief advance claimed for the skin test is that it will confirm the diagnosis during the first week of the disease when serum agglutinins are always absent.

Moss and Weilbaecher have employed animal inoculation as a means of diagnosis in obscure cases. These investigators have found the method useful in septicemia and in severe fulminating cases, in which agglutinins may be late in appearing or may be absent throughout the illness. They also report lung puncture as a satisfactory method of obtaining material for animal inoculation in cases of atypical pneumonia in which tularemia with pneumonic involvement is suspected. Animal inoculation and culture of the organism are too technical for the average physician but they should be kept in mind as useful methods of diagnosis in obscure cases.

Of most importance is the suspicion that the disease is present. Most patients do not mention the primary lesion nor do they volunteer information regarding the recent handling of rabbits or removing a tick from any part of the body. Many are unfamiliar with such sources. Repetition of the agglutination test at weekly intervals or oftener will frequently be repaid by a positive test and indicate the correct diagnosis. A titer of 1:80 is usually considered diagnostic and a rising titer, even if low in the beginning, is also diagnostic.

TREATMENT

A great many cases are of such a mild nature that symptomatic and supportive treatment alone is adequate. Many different drugs have been utilized in the therapy of tularemia with considerable success, including neoarsphenamine, mercurochrome, iodides, quinine, acriflavine and metaphen to mention just a few. This is probably accounted for by the fact that tularemia is a disease of low mortality, about five percent, and high morbidity. There are undoubtedly many cases that recover spontaneously without ever having the correct diagnosis established. Many mild cases never consult a physician.

Lee Foshay of Cincinnati has developed specific serum therapy which is probably the most scientific advance in treating tularemia. He has studied the effect of serum on both mortality and morbidity with adequate controls. He reports a mortality rate of six percent in 518 unselected untreated cases, used as controls, while in 600 unselected treated cases the rate was 2.2 percent. The morbidity was likewise appreciably reduced.

Several observers have used sulphanilamide without influencing the course of the disease. Moss and Weilbaecher have recently reported striking improvement following the administration of sulphathiazole. It is hoped that this drug will influence tularemia in much the same way as it has pneumonia, and if these observations can be confirmed it will simplify the treatment so that it will be readily available to all. They administered two grams of the drug every three hours for the first two or three doses and then maintained a high blood level by giving one gram every three hours thereafter.

At the present time the early administration of adequate doses of antitularense serum appears to be the best form of treatment. This is especially true in the typhoid type and tularemic pneumonia in which the highest mortality occurs. The usual dose is 30-60 cc given intravenously and repeated in 72 hours if necessary. Serum sickness is very common, about 50 percent, and according to Foshay, severe in 16 percent.

A simple antiseptic dressing, such as four percent xeroform ointment, should be applied to the primary lesion and otherwise left alone. It should never be incised. Injudicious treatment of this nature may precipitate fatal septicemia. Suppurative lymphadenopathies should be incised and drained when softening has occurred, not before. Most cases show rapid improvement when this happens.

The present report covers ten cases, nine of the ulcero-glandular, and one typhoid type, observed between June, 1937 and November, 1940. All recovered. One case followed tick bite, eight handled wild rabbits, and one had no definite source. Nine had a primary lesion at the time of treatment or gave such a history. The diagnosis was confirmed in each instance by positive agglutination tests made after the first week of illness. Intradermal tests were not used. Two patients had developed large pleural effusions when first seen, but pneumonia was not observed. The first complained of fever, cough, shortness of breath and loss of weight of three weeks duration and came for examination to confirm a previous impression of tuberculosis. When his chest was bared for examination an ulcer about .5 cm in diameter was noted in the right axillary region. The lymph nodes were enlarged and tender. When questioned about this he stated that three weeks previously he had removed a wood tick from that region but had not associated it with his pulmonary complaints. The agglutination test was strongly positive. All symptoms cleared completely under symptomatic treatment. The second came because of shortness of breath which had been present about two weeks. Examination revealed a massive effusion in the left pleural cavity. The fluid was clear, amber colored and contained no cells. She volunteered the information that a week before respiratory difficulty appeared, she had had sores on both thumbs and admitted skinning wild rabbits a few days before the primary lesion was noticed. She likewise made a complete recovery under symptomatic treatment.

The diagnosis was made in one case of fever by means of the agglutination test. Recovery was prolonged and tedious. This case was placed in the typhoid group.

Six of the ten cases received .6 gm neoarsphenamine at weekly intervals with good results in five. An average of five doses was given. Two cases received sulphanilimide without any apparent benefit. One patient did not respond to neo-arsphenamine or sulphanilimide but continued on a low grade febrile course for over six months, this in spite of the fact that the regional lymph nodes suppurated, were incised and drained, and healing had occurred rather early. Apparently a transition from the ulcero-glandular to the typhoid type occurred. This patient would probably have received benefit from serum. One patient came for drainage of an axillary abscess. The primary lesion had since healed and forgotten about. The agglutination test was reported positive in all dilutions. The administration of serum as well as neo-arsphenamine must be early in the disease to derive the greatest benefit.

CONCLUSIONS

1. Tularemia is a wide spread disease showing a recent increase in reported cases.

This increase is noted along with a recent increase in the number of wild rabbits in this region.

2. Tularemia assumes bizarre characteristics at times but the majority fall into the ulcero-glandular class.

3. Tularemia should be kept in mind in obscure fevers and unexplained pneumonias. The diagnostic tests are discussed and the importance of obtaining a reliable history to exposure emphasized.

4. Specific antiserum seems to be the best method of treatment especially in the severe cases. Chemotherapy has not had adequate trial. The first reports are very encouraging regarding sulphathiazole and discouraging regarding sulphanilimide.

5. Ten cases are reported of which six received neo-arsphenamine with good results in five.

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The Diagnosis and Control of Brucellosis*

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Brucellosis is an infection having no pathognomonic symptoms or signs. Moreover, patients in the United States rarely appear dangerously ill; hence the taking of a detailed history and the performance of a complete physical examination are readily neglected. In addition, many practitioners have not been familiar with the nature of this infection, nor are they accustomed to consider it in differential diagnosis. It is for these reasons that admittedly erroneous diagnoses have been made in so many cases. It has been gratifying, however, to see the accuracy with which brucellosis was diagnosed once the physician had seen cases or had familiarized himself with the clinical characteristics of the disease.

When the disease has been running for weeks and a temperature chart is available, the diagnosis is easy if the possibility of brucellosis is kept in mind. It is in the first weeks that difficulties arise. A list of the diseases for which brucellosis has been mistaken would include practically all the affections accompanied by a rise of temperature. The clinical symptoms, although suggestive, can never be conclusive.

The importance of laboratory tests in diagnosis has been stated repeatedly. The laboratory procedures which are of value in diagnosis are the agglutination test, the Brucellergen intradermal test, cultural studies, white blood cell and differential counts, and opsonocytophagic tests. The two of the greatest value in differential diagnosis are the intradermal and the agglutination tests. They are usually readily available, often without cost to the patient or physician. It may be urged, therefore, that these should be more frequently used in the investigation of febrile illnesses. Agglutinins can usually be demonstrated when the patient first applies for medical advice in infections with an insidious onset, while in those with sudden onset they may not appear until the end of the second week or, according to Simpson occasionally not until the fourth week. Apparently it is true of brucellosis as of typhoid that infrequently the serum of an infected individual may persistently fail to show any agglutinins. It must always be remembered also that a positive agglutination test may be related to the past or subclinical infection and not the present ailment of the patient. Blood, urine, and stool cultures are all valuable in the study of suspected cases of brucellosis, but these are practical in diagnosis only when the patient is within easy reach of a laboratory. Any culture study in this infection will consume at least one week, and a negative report on a blood culture cannot be made reliable until the end of the third or fourth week. Moreover, negative cultural findings on one examination can scarcely be given any weight. Cultural studies are, therefore, limited in their applicability as a diagnostic test.

The white blood cell findings in brucellosis are in no way peculiar to this infection; still a leucopenia with a relative lymphocytosis will serve to rule out all but a few conditions with which this disease may be confused.

The agglutination and complement-fixation tests in their applications to *Brucella* infections have been exhaustively studied by many investigators in Europe and North America. Their value and limitations are now well established. The complement-fixation test as a routine procedure in detecting

*Read before the Public Health Meeting, Annual Session, Oklahoma State Medical Association, May 20, 1941, in Oklahoma City.

TABLE I.

The Comparative Results of Diagnostic Tests on 100 Unselected Cases of Human Brucellosis

Total No. Cases	AGGLUTINATION TITER		OPSONIC TEST		BRUCELLERGEN TEST	
	No. 1:50 or Above	No. 1:25 or Negative	No. Positive	No. Negative	No. Positive	No. Negative
100	25	75	94	6	98	2

Brucella infection has fallen into disuse because of the time and attention that are required for its operation and because it does not furnish more information than the agglutination test, which is simpler to perform and gives results just as dependable.

Two methods of performing the agglutination test to detect *Brucella* infection in human beings and animals are available: the test-tube method and the rapid method. Both have been studied critically by many workers and found satisfactory.

Brucella agglutinins as a rule do not appear in the blood until ten days after the onset of the disease. Occasionally they cannot be detected during the course of the disease. This is especially true of the chronic form. It has been the author's observation, from a comparative study of the disease on the Island of Malta and in the United States, that agglutinins are found more often in the *Br. Melitensis* type of infection than in the *Br. abortus* type. Very few, if any, *Br. melitensis* cases fail to show agglutinins during the course of the disease.

A serum agglutination titer of 1:50 or higher, obtained on a patient showing clinical evidence of infection, should be considered as presumptive evidence of active *Brucella* infection. One should rely as much on the history of the case and other confirmatory tests as on the agglutination test in detecting active *Brucella* infection in human beings.

If the serum agglutination titer of an individual, following the disappearance of all clinical symptoms and signs of the disease, is found to be slowly decreasing as shown by a repetition of tests at 30 to 60 day intervals, it is evidence of recovery from infection.

The agglutination test, which has played such an important role in the diagnosis of animal infections, is of limited value in the chronic form of human brucellosis. The writer and his associates have had the opportunity to study the value of the various diagnostic tests on 150 brucellosis patients a year for the past three years that were sent to the laboratory by physicians. Most of the cases were of the chronic form. The agglutination test was of a sufficient titer in only 25 percent of those examined to indicate active infection (see Table I).

The results of the agglutination test may also be misleading because individuals are often found to have *Brucella* agglutinins in their blood serum when they show no symptoms of *Brucella* infection. These are chiefly laboratory workers, practicing veterinarians, farmers, and packing-house employees. By way of illustration let us consider the status of veterinarians with respect to the disease. Those who are engaged in cattle practice come in contact with *Br. abortus* to a greater extent than any other single group of people. It has already been shown by serological tests made on veterinarians in Europe and this country that a considerable percentage have *Brucella* agglutinins in varying degrees in their blood. While very few of them give a history of having a clinical course of the disease, many report the occurrence of skin eruptions and malaise after removing retained placentae from aborting cows. The malaise is usually characterized by dullness, headache, sweating and aching in the muscles and joints. There may occur an elevation of the temperature. These symptoms may occur from contact with the diseased tissues or from the ingestion or inhalation of the organism alive or dead, or from the ingestion of sterile broth filtrates on which the organism has grown. It is necessary, of course, that the specific allergen pass through the epithelium of the skin, the respiratory tract or the digestive tract. Individuals in the general population as well as those in particular groups may show the symptoms just mentioned when exposed to *Brucella*.

Cross agglutination between *Brucella* antigen and those for other disease has been the subject of many papers, but only in the case of tularemia is there likely to arise confusion as to the interpretation of the test. Francis and Evans have observed that cases of tularemia may show agglutinins in a low titer for *Brucella*. In personal communications the author has been informed of two other cases of tularemia in which the blood serum showed agglutinins for *Brucella* as well as *Pasteurella tularensis*. Both cases gave a negative skin reaction.

Since the widespread occurrence of undulant fever in man in the United States was established, considerable attention has been given to the diagnosis of the disease by

TABLE II.

Comparison of the Results of the Agglutination Test with Results of Other Laboratory Tests on 41 Clinical and 49 Subclinical Cases of Brucellosis

Total No.	Clinical Cases 41				Sub-clinical Cases 49			
Maximum Agglutination	Neg.	1:25	1:50	1:100 or Higher	Neg.	1:25	1:50	1:100 or Higher
Titers, No.	4	12	7	18	26	8	6	9
Blood Culture, Positive	3	12	7	13	1	0	1	1
Brucellergen Test, Negative	0	0	0	0	1	1	1	0
Brucellergen Test, Positive	4	12	7	18	25	7	5	9
Phagocytosis, Low or Moderate	3	10	6	14	16	6	4	7
Phagocytosis, Marked	1	2	1	4	0	1	1	2
Phagocytosis, Negative	0	6	0	0	10	1	1	0

allergic methods. Data have accumulated which show that this method when used alone will only detect Brucella allergy. It will not differentiate between post and active infection.

Many physicians often observe the occurrence of a symptom complex in patients not unlike that of acute or chronic undulant fever, but are confused in making a diagnosis because the serum reaction to an agglutination test for *Brucella* infection is negative. If in addition to the agglutination test an intradermal test is performed with a suitable agent and a positive reaction is obtained, this evidence alone is not sufficient to warrant a diagnosis of undulant fever, because all individuals who have been infected with *Brucella* in the past as well as those who are actively infected will show a skin reaction to a satisfactory *Brucella* allergen.

While making a study of methods for interpreting a positive Brucellergen test obtained in humans, the author and his associates discovered that the neutrophilic leucocytes in whole citrated blood of human beings who had recovered from brucellosis phagocytized *Brucella* cells in large numbers in a proper phagocytic system. It was also observed that the same type of leucocytes in whole blood from actively infected cases showed a lower degree of phagocytic activity than that seen in individuals after recovery and in the case of those who presented no past or present history of infection, little if any degree of phagocytosis was observed. The first two groups show a positive skin test, while the third group does not.

It has been the observation of the author that if a positive skin reaction is obtained in doubtful cases of brucellosis, the positive reaction does not have specific significance until a determination of the opsonocytophagic power of the whole blood is made. The latter test should be made at the same time or within several days after the skin test is performed, because the allergen may cause an agglutinin and opsonin response.

The procedure in performing the *Brucella* opsonic test and the system of interpreting the results may be found elsewhere.

During an epidemic of melitensis brucellosis a short time ago, an opportunity was presented for making a comparative study of four diagnostic tests on 41 clinical cases, and 49 classified as subclinical. The results of this study are set forth in Table II.

The results of the blood cultures, the skin test, and phagocytosis test on each case are grouped together according to their maximum agglutination titers. Of the four clinical cases whose blood sera were negative to the agglutination test, three showed a positive blood culture on the same date. The blood examinations in two of the four cases were made ten days and seven days after onset and in one, three days before the onset. The blood culture was positive in 12 other clinical cases which at the time showed a maximum agglutination titer of 1:25. Data showing the interval between the date of onset and the date the agglutination test was made and titer obtained are set forth in Table III. The intervals varied from one to 80 days. There does not appear to be any relationship between the strength of the titer and the time the test was made after onset.

The Brucellergen skin test was positive in all clinical cases. The size of the local reaction varied from two to six cm. The results of the phagocytosis test were confusing in eight of the 41 clinical cases in that the phagocytic picture was similar to that observed in immune individuals. These results agree in many respects with the results of the phagocytic tests conducted on *Brucella melitensis* infected individuals on

the Island of Malta. In many cases of *Brucella melitensis* infection, most of the leukocytes in the patients' blood in a phagocytic system showed a marked phagocytosis of *Brucella* cells. It is obvious from the results of each of the laboratory tests, that one cannot place too much reliance on any one test to confirm the early diagnosis of clinical brucellosis. The results of all the available tests taken together must be carefully analyzed in arriving at a positive diagnosis.

TABLE III.

The Results of the Agglutination Test And Blood Culture on 41 Clinical Cases Of Brucellosis

Case No.	Date of Onset	Date of Tests	Blood Culture	Maximum Agglutination Titer
1	12-10-38	12-20-38	+	negative
2	12-19-38	1-30-39	+	+1:25
3	12-26-38	3-17-39	not taken	+1:500
4	12-27-38	1-7-39	+	+1:200
5	12-27-38	2-1-39	+	+1:50
6	12-28-38	1-13-39	+	+1:500
7	12-28-38	1-17-39	+	+1:50
8	12-28-38	1-27-39	—	+1:500
9	12-28-38	1-17-39	+	+1:50
10	12-28-38	1-12-39	—	+1:100
11	1-5-39	1-13-39	+	+1:50
12	1-5-39	1-23-39	+	+1:100
13	1-5-39	1-24-39	+	+1:500
14	1-6-39	1-28-39	+	+1:200
15	1-6-39	1-13-39	+	negative
16	1-6-39	1-12-39	+	+1:25
17	1-6-39	1-14-39	+	+1:100
18	1-6-39	1-9-39	+	+1:50
19	1-7-39	1-12-39	+	+1:25
20	1-7-39	1-23-39	+	+1:500
21	1-8-39	2-1-39	+	+1:500
22	1-9-39	1-13-39	+	+1:25
23	1-10-39	2-14-39	+	+1:100
24	1-10-39	1-19-39	+	+1:25
25	1-12-39	1-14-39	+	+1:25
26	1-13-39	1-14-39	+	+1:25
27	1-13-39	1-20-39	+	+1:25
28	1-13-39	1-24-39	+	+1:50
29	1-15-39	1-19-39	+	+1:50
30	1-16-39	1-19-39	+	+1:25
31	1-19-39	1-24-39	+	negative
32	1-20-39	1-24-39	+	+1:25
33	1-20-39	1-25-39	+	+1:50
34	1-23-39	1-24-39	+	+1:100
35	1-25-39	2-9-39	—	+1:500
36	1-30-39	1-27-39	—	negative
37	2-1-39	2-18-39	+	+1:200
38	2-3-39	2-1-39	+	+1:500
39	2-5-39	2-1-39	+	+1:25
40	2-7-39	2-6-39	—	+1:100
41	2-10-39	2-13-39	+	+1:25

+ = complete agglutination or positive blood culture

— = no agglutination

In considering the results on the group classified as subclinical cases, one must take into consideration the possibility that many of them may have been exposed to infection several months or years before the study was made. The results of laboratory tests conducted on groups of people during the past several years reveal that from 10 to 15 percent may have at one time been infected with *Brucella*. The results of the phagocytic test, however, in which only four of the subclinical group show a high phagocytosis, would indicate that most of them were exposed to infective material on or near the same date as the clinical cases.

It is interesting to note that a positive blood culture was obtained from each of three of the case which failed to show the slightest symptoms of the disease before or after culturing. The three cases in question showed a positive skin test and moderate degree of phagocytosis at the time the blood cultures were taken. The skin test was negative in three of the subclinical cases. Two of these showed specific agglutinins at the time of the skin test.

The data set forth in Table II show conclusively that it is possible to demonstrate active infection long after exposure to infective material in individuals who never develop clinical manifestations of brucellosis. In other words, active infection in the case of brucellosis does not necessarily imply clinical infection.

Clinical laboratories and investigators have encountered cases in which the leucocytes in whole blood were found to show a considerable degree of phagocytic activity, but at the same time the Brucellergen test was negative. Two such cases have been brought to the attention of the author.

There are two possible causes to which one can attribute the absence of Brucellergen skin allergy in a patient whose blood shows a high degree of opsonic activity for *Brucella*. One is the presence of opsonins in the blood for *Brucella* in those who have recovered from or are actively infected with *B. tularensis*. Two such cases have been brought to the attention of the author. The other is due to the failure of a small percentage of patients infected with *Brucella* to develop skin allergy and show a positive reaction to Brucellergen or other *Brucella* allegic agents.

CONTROL OF BRUCELLOSIS

A paradoxical situation exists at the present time in the control of brucellosis in that there is sufficient knowledge available on the

nature of brucellosis effectively to suppress its spread in animals, and from animals to man. The important hosts of *Brucella* are known, the routes through which the organism is eliminated from the hosts have been determined, and the diagnostic procedures have been perfected to a high degree of accuracy. But despite all this usable knowledge the number of reported human cases continues to increase. The sanitarian is confronted with a perplexing problem of applying control measures effectively without affecting too seriously the economic interests involved.

Since brucellosis is primarily an animal borne disease it is exiomatic that the reduction of its incidence in man can only be brought about by either suppressing the disease in animals or by effectively breaking the infectious chains between animals and man.

Before effective control measures can be considered and put into practice in a given region, it is essential to know (1) the animal hosts, (2) the species of *Brucella* involved, (3) the means by which the organism is conveyed from animals to man. (See Table IV).

If the incidence of brucellosis is found to be high in more than one species of animal, and more than one species of *Brucella* is involved, considerable difficulty will be encountered in formulating control measures that will be applicable to all animals alike. The task becomes complex when attempts are made to prevent the transmission of the disease from one species of animal to another and from more than one animal host to humans.

If only one species of *Brucella* is found present in a given region, the problem of control is greatly simplified, but if more than one is implicated, those measures taken to suppress the dissemination of one species of *Brucella* may be unworkable for another species.

The three control measures that have been advocated and used in animal brucellosis are: (1) slaughter of infected animals, (2) segregation of infected animals from the non-infected on separate farms, (3) immunization of calves under eight months of age.

The slaughter plan for removing infected animals with the expectation of eliminating brucellosis in cattle has been in operation in certain states for 15 years, and on an extensive scale under federal supervision in most of the states for the past six years. The plan was placed in operation with a great deal of enthusiasm and has received the support of the veterinary profession and breeders' organizations. The slaughter plan was instituted in the face of the fact that previous preliminary surveys had indicated the

TABLE IV.

Viability of *Brucella* Outside the Animal Body

Material	Length of Life
Gastric juice	2 hours
Hog Cholera virus	5 months
Infected bovine fetus	7 months
Infective milk (cow)	10 days
Ice cream	30 days
Butter	142 days
Roquefort cheese	2 months
Urine (bovine)	4 days
Hog spleen (-10°F)	30 days
Hog spleen (in brine)	40 days
Mosquitoes (exposed)	48 hours
Flies (exposed)	120 hours
Soil (dry)	27 days
Soil (moist)	37 days
Tap water (25°C)	77 days
Tap water (-40°C)	114 days
Sunlight	4½ hours

incidence of infection in cattle would average 15 percent and with the knowledge that the cow is not the only host for *Brucella*. The failure of several enthusiastic advocates of the plan to give any consideration whatsoever to sources of infection other than the infected cow, and the incubation period in the disease has accounted for its failure in many instances and caused much disappointment in those directly concerned.

An unbiased analysis of the records of the cooperative federal-state Bang's disease program shows that considerable progress has been made in reducing the percentage of infected cattle and, further, the disease has been eliminated in a large number of herds. The success of the slaughter plan in eliminating the disease from infected herds appears to be inversely proportional to the size of the herd. Difficulty has been encountered in eliminating the disease from herds of a considerable size and preventing reinfections in many smaller ones. The failures in most instances cannot be attributed to laxity in sanitary practices or inadequate supervision. It is possible that when more information is available concerning the incubation period of the disease in the cow and more consideration is given to other hosts of *Brucella*, less difficulty will be met in the elimination of brucellosis from large herds.

In certain regions the milch goat or the hog is the chief reservoir for the source of the disease in man. It follows that the elimination of the disease in the cow may have

little, if any effect on the lowering of the incidence of brucellosis in humans. The effectiveness of the slaughter method in infected goats and hogs is yet to be determined.

Control of the disease in cattle by means of calf vaccination is worthy of trial. Experimental data in support of vaccination of calves with a live culture of low virulence was first presented by Cotton and Buck and Smith in 1934. Since 1934, a considerable amount of data have been collected under experimental and natural conditions by several investigators which support the original findings. A high degree of protection against infection appears to be obtained in 70 percent of the vaccinated calves. The immunity appears to persist for one or more years. There have been no data obtained which indicate that the live bacteria in the vaccine is infectious for the calf under eight months of age. Vaccination of calves would in time result in the rearing of herds of cattle free from brucellosis.

There is another simple and inexpensive measure which could be taken in to prevent the spread of the disease in animals and thereby aid in keeping it under control. It is the requirement of a negative blood tes. on animals sold for breeding or for dairy purposes. If the sale of infected animals from one farm to another, or from stockyard to farm is prevented, the spread of the disease to farms where infection does not exist can be minimized. Such a measure should not be too great a burden for the farmer to bear. It would be to his interest to support and to cooperate in making the plan a success.

There still seems to exist much confusion and guessing regarding the necessary hygienic and sanitary measures that should be taken to prevent the transmission of brucellosis to humans. Today there is as much knowledge, available on the habits of *Brucella*, the channels through which they are eliminated from hosts and the means of conveyance to humans as any other pathogenic bacteria.

The incidence of the disease in humans could quickly be reduced to a minimum if the owners of livestock were taught to use more caution in the handling of infected animals, their excreta and secretions; and by the proper pasteurization of all milk from infected animals used for human consumption.

When the present knowledge is made use of to the fullest extent by sanitarians and those intrusted with the guarding of human health, brucellosis in man will be a disease of the past instead of a disease of the future.

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• THE PRESIDENT'S PAGE •

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EDITORIALS

PROGRAM FOR THE STATE MEETING

Elsewhere in the Journal you will find a complete outline of the program to be presented at the 50th Annual Meeting of the Oklahoma State Medical Association.

To some of you, the departure from the conventional type of program presented in the past will be a welcome change. To others, it will appear as though the activities of the various sections which make up the scientific program have been deliberately and maliciously curtailed. To still others, the idea may seem a novel experiment worthy of trial and consideration for future meetings.

Since its inception, the Scientific Work Committee has had but one aim and one goal. This is to present a high type of scientific program which will be interesting and valuable to the entire membership. Following the requests of the various sections, we have provided guest speakers for each section again this year as was done last year. The Committee felt, furthermore, that the membership should be taken into consideration in choosing subjects and material for presentation. As a result, a questionnaire was sent to each member, and we are incorporating those subjects in the program which received the largest number of requests. We extend a hearty vote of thanks to the individuals who responded to our questionnaire.

Your committee has been faced with a difficult task this year in providing guest speakers. We have also been confronted with the problem of providing a meeting place where nine sections could meet at the same time. We must admit there is no hotel or available building in Oklahoma large enough to accomodate a meeting of this size. We have made no deliberate attempt to curtail the activity of the respective sections, but it is a physical impossibility to provide each one with a suitable meeting place without subjecting the Association to the prohibitive expense of building section rooms on the floor of the Coliseum. This is the reason we have adopted the program as presented herewith.

The round-table discussion of Sulfanilamide Therapy to be participated in by all of the guest speakers, as well as the audience, is an innovation this year, and it is hoped that it will be a very valuable and instructive presentation.

Whether or not you agree with the arrangement of the program, we request your indulgence and cooperation. If there ever was a time in the history of our profession when it is essential for us, as individuals, to get behind and support organized medicine to the fullest extent, that time is now. American medicine will necessarily have to assume world leadership at the termination of the war. It behooves all of us, therefore, to become actively interested and lend full support to the component units of organized medicine which are the County and State Medical Societies. We urge you to attend this meeting, and we are confident that you will find something of interest and value in the papers presented by our essayists.—C. R. R.

MEDICINE PLUS

In this hour of trouble, embracing a world-wide war of unprecedented gravity, every good doctor must not only preserve his own equanimity, but with incredible self-control, he must pass mental poise to those who are less stable. Calm courageous attitudes while in action or in time of suspense, must become universal, if we want to win the war and preserve our democratic way of life. Otherwise, the psychologically unstable will become incapable of making constructive contributions toward the solution of our national problems. Failing in this, they not only retard progress by their own impotency, but they become a burden upon those who must carry on in spite of all obstacles.

In the history of mankind, there has never been a time when the penalties of war descended upon society in such a deadly, comprehensive fashion. Modern warfare spares no human being, not even the prenatal. Lying-in hospitals have been bombed; the sanctity of home has no security; the accumulated treasures of art are being ground into dust.

Our men on the far-flung battle front must not only meet their foes face to face, but they must fight with the deadly fear that their loved ones at home may be the innocent victims of unannounced bombing or barbarous invasion. Though our men stand at the front, they too must await news of the war.

This brief comment shows what an important bearing modern warfare has upon mass psychology. Never before has there been such valid reason for universal mental strain. Already 50 percent of the hospital

beds in this country are occupied by mental cases, and psychiatric deficiencies stand fifth as a cause for rejection of registrants. But doctors know it is doubtful if this high incidence of nervous and mental disease really represents a genuine decline in mental stability. It seems more reasonable to attribute the increasing prevalence of psychiatric difficulties to the trying task of adjustment in a swiftly moving, ever-changing new way of life. Unfortunately this adjustment had to be integrated with our recovery from the strain of the last world war, with its untold heroisms and self-sacrifices which, "for the safety of democracy," poured out the blood of ten million people.

If, perchance, we wonder what a doctor can do to help win a sacred cause in a world of strife and confusion, let us remember that his first duty is to keep his head clear and his hands ready for the immediate task. In so doing, he can help stabilize mass psychology and possibly stave off the evil effects of widespread hysteria, which would inevitably lead to certain defeat.

With the above in mind, doctors must help people realize that it is not necessary to draw a fine line between normal and abnormal behavior, but that it is essential for each individual to become a living example for those about him. This is no time to ask, "am I my brother's keeper?" There is both sanity and safety in service. We must do what we can to make society fool-proof. Concerted effort in this direction will do more for the fearful, wavering mind than all the medicine in the world.

Our hospitals for nervous and mental diseases are full, doctors are being called into government service, and those who remain at home are over-worked. But this is no time for us to sink into the "slough of despond." On the contrary, our fortitude and composure must serve as an antidote for those who have an idiosyncrasy toward calamity. While threatened with a lethal dose of melancholy or fear, they will need our sympathy and support. The survival of democracy will depend upon our composite response to the great emergency now claiming our attention. This emergency can be met successfully only through the elevation of our national zeal to a high average of stability and continuity. —L. J. M.

Dr. Vernon Speaks at Arkansas Meeting

Dr. W. C. Vernon of Okmulgee was guest speaker at the meeting of the Sebastian County Medical society of Arkansas, February 19, in Fort Smith, Ark.

Doctor Vernon chose "The Injection Treatment of Hemorrhoids" as the subject of his talk.

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PROGRAM

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GREETINGS FROM THE TULSA COUNTY SOCIETY

Every other year the Tulsa County Medical Society shares honors with the Oklahoma County Society in acting as host society to the annual meeting of the Oklahoma State Medical Association. This year the Tulsa Society automatically becomes host on April 22, 23 and 24.

The Tulsa Society is alert and fully aware of its obligations when it accepts the honor as host. It shall be our duty to see that every detail is carried out in the execution of the main purpose of the meeting—the scientific program. The entertainment and side lights incident to the program shall be our responsibility to the extent that everyone in attendance will feel fully compensated for whatever sacrifice he may have made to be our guest.

In order that all events as planned shall be carried out on schedule and without detraction by holding two events at the same time, the Tulsa County Society has arranged for a special **Buffet Supper** at the Mayo Hotel at 6:30 p.m. on Tuesday evening, April 21. Every member of the State Medical Society is cordially invited to this affair, as guests of the local society. The supper is to be followed by two interesting speakers on medico-legal problems. If you find it possible to attend this supper, the Tulsa County Executive Secretary would appreciate a post card to that effect.

The Women's Auxiliary of our local society has made plans for entertainment of the women and they join in this formal invitation to Tulsa. Make your plans to bring your wife with you.

We shall look for you on Tuesday evening, April 21. The Buffet Supper will be a stag affair.

H. B. Stewart, M.D., President,
Tulsa County Medical Society.

General Information

HEADQUARTERS
Coliseum, Tulsa

REGISTRATION
Coliseum

All physicians except those from outside the state, visiting guests, and those on military assignment, must have membership cards for 1942 before registering. Dues will not be accepted at the registration desk except from County Secretaries.

GENERAL SESSIONS AND SECTION MEETINGS

All meetings will be held in the main foyer of the Coliseum. Section meetings will start at 9:00 A. M., and the General Sessions at 2:30 P. M.

GUEST SPEAKERS

Charles C. Dennie, M.D., Professor of Clinical Dermatology, University of Kansas School of Medicine, Kansas City, Mo.

George Herrmann, M.D., Professor of Clinical Medicine, University of Texas School of Medicine, Galveston, Tex.

John C. Burch, M.D., Associate Professor of Obstetrics and Gynecology, Vanderbilt University, School of Medicine, Nashville, Tenn.

T. Leon Howard, M.D., Associate Professor of Surgery (Urol.), University of Colorado School of Medicine, Denver, Colo.

Titus H. Harris, M.D., Professor of Neurology and Psychiatry, University of Texas School of Medicine, Galveston, Tex.

M. Edward Davis, M.D., Associate Professor of Obstetrics and Gynecology, University of Chicago, the School of Medicine, Chicago, Ill.

A. B. Reese, M.D., Assistant Clinical Professor of Ophthalmology, Columbia University College of Physicians and Surgeons, New York City, N. Y.

Robert L. Sanders, M.D., Associate Professor of Surgery, University of Tennessee College of Medicine, Memphis, Tenn.

Hugh L. Dwyer, M.D., Professor of Clinical Pediatrics, University of Kansas School of Medicine, Kansas City, Mo.

HOUSE OF DELEGATES

The House of Delegates will meet Wednesday at 8:00 P. M. and at 8:00 A. M., Thursday, in the Ivory Room, at the Mayo Hotel.

COUNCIL

The Council will meet Tuesday evening, April 21, immediately following the Buffet Supper of the Tulsa County Medical Society, in the French Room, Mezzanine floor, Mayo Hotel, and thereafter on the call of the President.

SCIENTIFIC ROUNDTABLE ON SULFONAMIDE THERAPY

An innovation for the Fiftieth Annual Meeting will be the conducting of a Scientific Roundtable on Sulfonamide Therapy, in which all of the guest speakers will participate. The Roundtable will be held in the Crystal Ballroom of the Mayo Hotel, Wednesday evening, at 8:00 o'clock. An opportunity will be afforded the audience to ask questions of the guest speakers.

WOMEN'S AUXILIARY

Registration will be in the lobby of the Mayo Hotel. The complete Program will be found on this page.

TECHNICAL AND SCIENTIFIC EXHIBITS

The exhibits will be displayed in the main foyer ofthe Coliseum.

RESOLUTIONS

Resolutions for the consideration of the House of Delegates should be prepared and submitted at the first meeting of the House of Delegates.

TULSA COUNTY MEDICAL SOCIETY BUFFET SUPPER

The Buffet Supper will be held Tuesday evening, April 21, in the Ivory Room of the Mayo Hotel at 7:00 P. M. All members of the Association and guests of the Association are to be the guests of the Tulsa County Medical Society. An attractive program on Medico-Legal and Economic subjects has been arranged.

OKLAHOMA UNIVERSITY MEDICAL ALUMNI LUNCHEON

The Luncheon will be held at 12:00, Thursday, April 23, at the Tulsa Club.

TEA FOR DOCTORS AND WIVES

The Tea will be held from 6:30 to 7:30, Thursday evening, April 23, in the Junior Ballroom, Mayo Hotel. Everyone is cordially invited.

PRESIDENT'S INAUGURAL DINNER DANCE

The Dinner Dance will immediately follow the Tea and will be held in the Crystal Ballroom, 16th floor, Mayo Hotel. Dr. Morris Fishbein, Editor of the Journal of the American Medical Association, will be the guest speaker.

GOLF TOURNAMENT

The Golf Tournament will be held at the Tulsa Country Club and will be Medal Play over 18 holes. Those wishing to compete in the Tournament may play any time during the Meeting. Green fee will be $1.00 Carl Simpson, M.D., Tulsa, is Chairman of the Tournament.

Women's Auxiliary Program

State Auxiliary Officers

President
Mrs. E. D. Greenberger
McAlester

President-Elect
Mrs. Frank L. Flack
Tulsa

Vice-President
Mrs. Rush Wright
Poteau

Secretary
Mrs. E. H. Shuller
McAlester

Treasurer
Mrs. L. S. Willour
McAlester

Historian
Mrs. A. R. Sugg
Ada

Parliamentarian
Mrs. W. A. Fowler
Norman

CONVENTION PROGRAM

Mrs. J. W. Childs
Convention Chairman

Wednesday, April 22, 1942

9:00 A. M.—Registration — Lobby, Mayo Hotel

7:30 P. M.—Pre-convention Executive Board Meeting, in the home of Mrs. J. W. Childs, 1616 South Madison, Tulsa.

Thursday, April 23, 1942

8:30 A. M.—Registration — Mezzanine, Mayo Hotel

10:00 A. M.—Annual Meeting — Parlor, Mezzanine, Mayo Hotel

1:00 P. M.—Luncheon — Marine Room, Mayo Hotel, $1.00
All visiting ladies invited. Tickets may be secured at the registration desk.

3:00 P. M.—Post-convention Executive Board Meeting, Parlor, Mezzanine, Mayo Hotel.

GUEST SPEAKERS

Annual Session, Oklahoma State Medical Association

Section on Dermatology and Radiology

Charles C. Dennie, M.D., Professor of Clinical Dermatology, University of Kansas School of Medicine, Kansas City, Mo.

Section on Medicine

George Herrmann, M.D., Professor of Clinical Medicine, University of Texas School of Medicine, Galveston, Texas.

Section on Obstetrics and Gynecology

John C. Burch, M.D., Associate Professor of Obstetrics and Gynecology, Vanderbilt University School of Medicine, Nashville, Tenn.

Section on Urology and Syphilology

T. Leon Howard, M.D., Assistant Professor of Surgery (Urol.), University of Colorado School of Medicine, Denver, Colo.

Section on Public Health

M. Edward Davis, M.D., Associate Professor of Obstetrics and Gynecology, University of Chicago, the School of Medicine, Chicago, Ill.

Section on Eye, Ear Nose and Throat

A. B. Reese, M.D., Assistant Clinical Professor of Ophthalmology, Columbia University College of Physicians and Surgeons, New York City, N. Y.

Section on General Surgery

Robert L. Sanders, M.D., Associate Professor of Surgery, University of Tennessee College of Medicine, Memphis, Tenn.

Section on Neurology, Psychiatry and Endocrinology

Titus H. Harris, M.D., Professor of Neurology and Psychiatry, University of Texas School of Medicine, Galveston, Tex.

Section on Pediatrics

Hugh L. Dwyer, M.D., Professor of Clinical Pediatrics, University of Kansas School of Medicine, Kansas City, Mo.

Scientific Program

Oklahoma State Medical Association
April 22, 23, 24, 1942
Coliseum, Tulsa

All Sections and Sessions will meet in the General Assembly Hall in the main foyer of the Coliseum.

Wednesday, April 22

General Chairman, C. R. Rountree, M.D., Oklahoma City

9:00 A. M.
SECTION ON DERMATOLOGY AND RADIOLOGY
Onis G. Hazel, M.D., Chairman

9:00 "Sarcoid of Boeck"—Onis G. Hazel, M.D., Oklahoma City, and Wayne M. Hull, M.D., Oklahoma City.
9:20 Discussion—John H. Lamb, M.D., Oklahoma City.
9:30 "Eczema—Modern Concepts of Treatment"—Harry Green, M.D., Tulsa.
9:50 Discussion—James Stevenson, M.D., Tulsa.
10:00 "The Treatment of Pregnant Syphilitic Women and the Results of Treatment"—Charles C. Dennie, M.D., Kansas City, Mo.

10:30 A. M.
SECTION ON GENERAL MEDICINE
W. W. Rucks, Jr., M.D., Chairman

10:30 "Hygiene in the Tropics"—Walker Morledge, M.D., Oklahoma City.
10:50 Discussion—Carroll M. Pounders, M.D., Oklahoma City.
11:00 "Atypical Pneumonia"—Samuel Goodman, M.D., Tulsa.
11:20 Discussion—Paul B. Cameron, M.D., Pryor.
11:30 "Exigencies of Cardiological Practice"—George Herrmann, M.D., Galveston, Tex.

12:00 Noon

1:00 P. M.
SECTION ON EYE, EAR, NOSE AND THROAT
F. Maxey Cooper, M.D., Chairman

1:00 "Relation of the Eye, Ear, Nose and Throat Specialist to the General Practitioner"—Marvin D. Henley, M.D., Tulsa.
1:20 Discussion—J. F. Gorrell, M.D., Tulsa.
1:30 "Causes of Blindness in Oklahoma"—Tullos O. Coston, M.D., Oklahoma City.
1:50 Discussion—J. A. Morrow, M.D., Oklahoma City.
2:00 "Office Therapeutics"—A. B. Reese, M.D., New York City, N. Y.

General Sessions

Chairman, Finis W. Ewing, M.D., President, Oklahoma State Medical Association

2:30 P. M.
OBSTETRICS AND GYNECOLOGY

2:30 "Physiological Approach to Gynecology"—John C. Burch, M.D., Nashville, Tenn.

3:15 P. M.
DERMATOLOGY AND RADIOLOGY

3:15 "Congenital Syphilis of the Bones and Joints of Infants"—Charles C. Dennie, M.D., Kansas City, Mo.

4:00 P. M.
PEDIATRICS

4:00 "Protection of Children Against Tuberculosis"—Hugh L. Dwyer, M.D., Kansas City, Mo.

Scientific Program

Thursday, April 23

General Chairman, T. H. McCarley, M.D., McAlester

All Sections and Sessions will meet in the General Assembly Hall in the main foyer of the Coliseum.

9:00 A. M.
SECTION ON OBSTETRICS AND GYNECOLOGY

Charles Ed White, M.D., Chairman

9:00 "Obstetrical Psychosis"—Bruce R. Hinson, M.D., Enid.
9:20 Discussion—Coyne Campbell, M.D., Oklahoma City.
9:30 "Delivering the Sick Woman"—E. P. Allen, M.D., Oklahoma City.
9:50 Discussion—J. B. Eskridge, Jr., M.D., Oklahoma City.
10:00 "The Etiology of Menstrual Disturbances"—John C. Burch, M.D., Nashville, Tenn.

10:30 A. M.
SECTION ON GENERAL SURGERY

A. Ray Wiley, M.D., Chairman

10:30 "Goiter"—C. C. Hoke, M.D., Tulsa.
10.50 Discussion—
11:00 "Treatment of Varicose Veins"—R. Q. Atchley, M.D., Tulsa.
11:20 Discussion—Carl J. Hotz, M.D., Tulsa.
11:30 "Carcinoma of the Colon: Lantern Slides"—Robert L. Sanders, M.D., Memphis, Tenn.

12:00 Noon

1:00 P. M.
SECTION ON PEDIATRICS

David J. Underwood, M.D., Chairman

1:00 "Scarlet Fever Immunization"—Hugh C. Graham, M.D., Tulsa.
1:20 Discussion—J. T. Bell, M.D., Oklahoma City.
1:30 "Problems of Behavior in Child Guidance"—Harold J. Binder, M.D., Oklahoma City.
1:50 Discussion—Carroll M. Pounders, M.D., Oklahoma City.
2:00 "Emergencies of the New-born Period"—Hugh L. Dwyer, M.D., Kansas City, Mo.

General Sessions

Chairman, C. R. Rountree, M.D., Oklahoma City

2:30 P. M.
General Medicine

2:30 "Functional Heart Disorders Including the Soldier's Heart"—George Herrmann, M.D., Galveston, Tex.

3:15 P. M.
Public Health

3:15 "The Endocrines in Obstetrics and Gynecology"—M. Edward Davis, M.D., Chicago, Ill.

4:00 P. M.
Eye, Ear, Nose and Throat

4:00 "Exophthalmos Associated With Thyroid Disease"—A. B. Reese, M.D., New York City, N. Y.

Scientific Program

Friday, April 24

General Chairman, R. C. Pigford, M.D., Tulsa

All Sections and Sessions will meet in the General Assembly Hall in the main foyer of the Coliseum.

9:00 A. M.
SECTION ON NEUROLOGY, PSYCHIATRY AND ENDOCRINOLOGY
Felix M. Adams, M.D., Chairman

9:00 "Recent Advances in Psychosomatic Medicine"—Charles E. Leonard, M.D., Oklahoma City.
9:20 Discussion—F. M. Adams, M.D., Vinita.
9:30 "Neuropsychiatric Aspects of Carbon Disulphide Poisoning with Report of Two Cases Occurring in Oklahoma"—M. D. Spottswood, M.D., Tulsa.
9:50 Discussion—James A. Willie, M.D., Oklahoma City.
10:00 "Complications Following the Use of Dilantin"—Titus H. Harris, M.D., Galveston, Tex.

10:30 A. M.
SECTION ON UROLOGY AND SYPHILOLOGY
Robert H. Akin, M.D., Chairman

10:30 "Urinary Lithiasis"—J. W. Rogers, M.D., Tulsa.
10:50 Discussion—Harry H. Hudson, M.D., Enid.
11:00 "Prostatic Resection and the Result Expected"—E. Halsell Fite, M.D., Muskogee.
11:20 Discussion—Robert H. Akin, M.D., Oklahoma City.
11:30 "Malignancy of the Bladder and Prostate with Reference to New Methods of Treatment"—T. Leon Howard, M.D., Denver, Colo.

12:00 Noon

1:00 P. M.
SECTION ON PUBLIC HEALTH
John A. Morrow, M.D., Chairman

1:00 "Ideals in Rural Obstetrics"—Isadore Dyer, M.D., Tahlequah.
1:20 Discussion—Edward N. Smith, M.D., Oklahoma City, and E. R. Muntz, M.D., Ada.
1:30 "Fluoroscopic Survey of Postnatal Syphilis in a Health Department Clinic—A Preliminary Report"—David V. Hudson, M.D., Tulsa, and S. C. Venable, M.D., Tulsa.
1:50 Discussion—E. A. Gillis, M.D., Oklahoma City.
2:00 "Practical Problems in Maternal and Child Health"—M. Edward Davis, M.D., Chicago, Ill.

General Sessions

Chairman, J. D. Osborn, M.D., Frederick

2:00 P. M.
Urology and Syphilology

2:30 "The Symptoms, the Diagnosis and the Treatment of Nephroptosis"—T. Leon Howard, M.D., Denver, Colo.

3:15 P. M.
Neurology, Psychiatry and Endocrinology

3:15 "Discussion of the Depressions as Seen in the General Practice of Medicine"—Titus H. Harris, M.D., Galveston, Tex.

4:00 P. M.
General Surgery

4:00 "Complications of Duodenal Ulcers; Their Surgical Management: Lantern Slides"—Robert L. Sanders, M.D., Memphis, Tenn.

MODIFYING NICOTINE INTAKE

NEED REQUIRE NO SACRIFICE OF SMOKING PLEASURE OR ECONOMY

EVERY physician knows the resistance of patients to any modification of smoking habits. Yet often it is desirable to reduce the intake of nicotine, the substance conceded to be the chief physiologic component of cigarette smoke.

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** J.A.M.A., 93:1110, Oct. 12, 1929*
Bruckner, Die Biochemie des Tabaks, 1936
*** The Military Surgeon, Vol. 89, No. 1, p. 7, July, 1941*

CAMEL

THE CIGARETTE OF COSTLIER TOBACCOS

ASSOCIATION ACTIVITIES

Association Takes Over Malpractice Insurance With London and Lancashire

The Tulsa County Medical society, which two years ago caused a drastic reduction in malpractice insurance rates for the doctors of Oklahoma by working out a group policy first with the Houston Fire and Casualty company and later with London and Lancashire, requested on February 10 that the State Association become the sole holder of the Master Policy and that all future control of the policy rest with the Association.

This action by Tulsa county was promptly accepted by the Council, and a new Master Policy has been issued in favor of the Association.

The work of the Tulsa County Medical society in bringing this saving on malpractice insurance to the entire membership of the Association has been one of the finest contributions to the Association that has ever been accomplished. On the basis of an average premium of thirty-five dollars, members of the Association have been saved approximately $17,500.00 a year.

Every member of the Association who carries malpractice insurance should cooperate in this program by carrying his insurance with London and Lancashire. Details concerning the policy may be obtained by writing the office of the Association.

Councilor District 10 Holds First Annual Meeting

The First Annual Meeting of members of the Tenth Councilor District of the Association was held on the evening of February 23, at Rock Gables near Hugo, with 24 members in attendance.

The meeting was called to order by Dr. Fred Switzer, Hugo, of the Choctaw County Medical society, and was then turned over to Dr. J. S. Fulton, Atoka, Councilor for the District. Doctor Fulton complimented the group on their attendance, and stated that such meetings should be continued in the future.

Dr. Finis W. Ewing, Muskogee, President of the Association, was introduced, and then he addressed the group on the subject of "Medicine and War." Doctor Ewing also spoke on the activities of the Association.

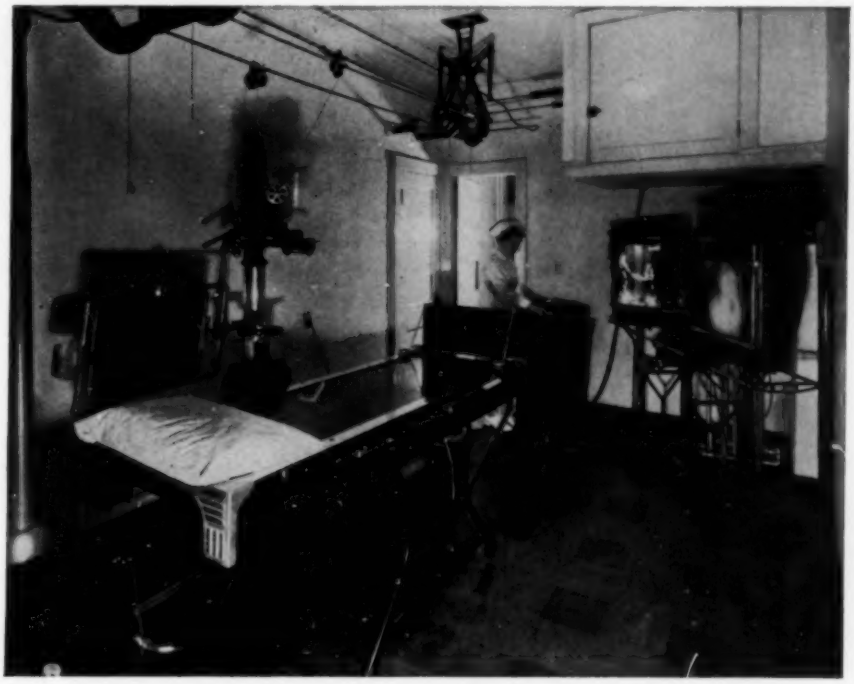
Major Louis H. Ritzhaupt, State Medical Officer, discussed "Selective Service and the Rehabilitation Program for Rejectees." After adjournment to hear President Roosevelt's radio address, the group heard a talk on "Procurement and Assignment" by R. H. Graham, Executive Secretary of the Association.

Delegates and Alternates to Annual Meeting Are Announced

In compliance with the by-laws of the Oklahoma State Medical Association, the delegates and alternates of the county societies who have been certified to the office by the local county societies are hereby announced.

All delegates and alternates will be seated in accordance with the membership certified as of March 22.

COUNTY	DELEGATE	ALTERNATE
Alfalfa	H. E. Huston, Cherokee	
Atoka-Coal	J. C. Canada, Atoka	W. W. Cotton, Atoka
	J. B. Clark, Coalgate	R. C. Henry, Coalgate
Beckham	H. K. Speed, Sayre	E. S. Kilpatrick, Elk City
Blaine	Virginia Olson Curtin, Watonga	L. R. Kirby, Okeene
Bryan	J. T. Colwick, Durant	J. A. Haynie, Durant
Caddo	E. W. Hawkins, Carnegie	G. E. Haslam, Anadarko
Canadian	J. T. Phelps, El Reno	M. E. Phelps, El Reno
Carter	G. E. Johnson, Ardmore	F. W. Boadway, Ardmore
	J. L. Cox, Ardmore	Walter Hardy, Ardmore
Cherokee	Robert K. McIntosh, Jr., Tahlequah	Isadore Dyer, Tahlequah
	Warren Beasley, Stilwell	
	William Newlin, Sallisaw	
Choctaw	E. O. Johnson, Hugo	O. R. Gregg, Hugo
Cleveland	F. C. Buffington, Norman	O. E. Howell, Norman
	Phil Haddock, Norman	W. B. Carroll, Norman
Comanche	O. L. Parsons, Lawton	Howard Angus, Lawton
Cotton	George A. Tallant, Walters	A .B. Holsted, Temple
Craig	F. M. Adams, Vinita	Lloyd H. McPike, Vinita
Creek	C. R. McDonald, Mannford	Charles Schrader, Bristow
Custer	McLain Rogers, Clinton	Gordon Williams, Weatherford
	Ellis Lamb, Clinton	Ross Deputy, Clinton
Garfield	V. R. Hamble, Enid	
	W. P. Neilson, Enid	
Garvin	Galvin L. Johnson, Pauls Valley	
Grady	W. H. Cook, Chickasha	
Grant	E. E. Lawson, Medford	
Greer	L. E. Pearson, Mangum	J. B. Hollis, Mangum
Harmon	W. G. Husband, Hollis	Russell Lynch, Hollis
Haskell	J. C. Rumley, Stigler	William S. Carson, Keota
Hughes	W. L. Taylor, Holdenville	
Jackson	E. S. Crow, Olustee	E. W. Mabry, Altus
Jefferson	L. L. Wade, Ryan	
Kay	J. C. Wagner, Ponca City	L. I. Wright, Blackwell
	Dewey Mathews, Tonkawa	G. S. Kreger, Tonkawa



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Many physicians have come to depend upon Polyclinic's X-Ray findings in the study of diseases of the bone, lungs, stomach and other conditions.

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Logan	L. A. Hahn, Guthrie	J. L. LeHew, Jr., Guthrie
Marshall	J. L. Holland, Madill	O. A. Cook, Madill
Mayes	Carl Puckett, Oklahoma City	V. D. Herrington, Pryor
McClain	O. O. Dawson, Wayne	G. S. Barger, Purcell
McCurtain	R. B. Oliver, Idabel	W. W. Williams, Idabel
McIntosh	Wm. A. Tolleson, Eufaula	D. E. Little, Eufaula
Murray	W. D. DeLay, Sulphur	F. E. Sadler, Sulphur
Muskogee	C. E. White, Muskogee	I. B. Oldham, Jr., Muskogee
	J. H. White, Muskogee	L. S. McAlister, Muskogee
Noble	A. M. Evans, Perry	T. F. Renfrow, Billings
Okfuskee	C. M. Cochran, Okemah	M. L. Whitney, Okemah
Oklahoma	W. F. Keller, Okla. City	W. W. Rucks, Jr., Okla. City
	Onis G. Hazel, Okla. City	Neil Woodward, Okla. City
	Walker Morledge, Okla. City	R. H. Akin, Okla. City
	L. C. McHenry, Okla. City	W. E. Eastland, Okla. City
	C. R. Rountree, Okla. City	M. F. Jacobs, Okla. City
	O. A. Watson, Okla. City	R. L. Murdoch, Okla. City
	J. B. Snow, Okla. City	R. L. Noell, Okla. City
	D. H. O'Donoghue, Okla. City	W. K. West, Okla. City
	C. M. Pounders, Okla. City	Harper Wright, Okla. City
	George Garrison, Okla. City	R. Q. Goodwin, Okla. City
	Allen Gibbs, Okla. City	George Kimball, Okla. City
Okmulgee	I. W. Bollinger, Henryetta	J. C. Matheney, Okmulgee
Osage	G. I. Walker, Hominy	Roscoe Walker, Pawhuska
Ottawa	M. A. Connell, Picher	M. M. DeArman, Miami
	Walter B. Sanger, Picher	F. L. Wormington, Miami
Pawnee	J. L. LeHew, Pawnee	R. E. Jones, Pawnee
Payne	A. B. Smith, Stillwater	R. E. Waggoner, Stillwater
Pittsburg	T. H. McCarley, McAlester	W. C. Wait, McAlester
	L. S. Willour, McAlester	Frank Baum, McAlester
Pontotoc	Ollie McBride, Ada	Sam A. McKeel, Ada
Pottawatomie	G. S. Baxter, Shawnee	E. E. Rice, Shawnee
	W. M. Gallaher, Shawnee	R. M. Anderson, Shawnee
Pushmataha	P. B. Rice, Antlers	E. S. Patterson, Antlers
Rogers	R. C. Meloy, Claremore	
Seminole	Claude S. Chambers, Seminole	Mack I. Shanholtz Wewoka
Stephens	C. N. Talley, Marlow	Claude B. Waters, Duncan
Texas	Johnny A. Blue, Guymon	Morris Smith, Guymon
Tillman	T. F. Spurgeon, Frederick	J. E. Childers, Tipton
Tulsa	R. A. McGill, Tulsa	E. Rankin Denny, Tulsa
	George Osborn, Tulsa	T. J. Hardman, Tulsa
	W. A. Showman, Tulsa	Eugene Wolff, Tulsa
	M. J. Searle, Tulsa	Carl Simpson, Tulsa
	Marvin D. Henley, Tulsa	W. A. Walker, Tulsa
	W. Albert Cook, Tulsa	W. R. Turnbow, Tulsa
	R. C. Pigford, Tulsa	M. D. Spottswood, Tulsa
	W. S. Larrabee, Tulsa	
Wagoner	H. K. Riddle, Coweta	J. H. Plunkett, Wagoner
Washington-Nowata	S. A. Lang, Nowata	K. D. Davis, Nowata
	L. D. Hudson, Dewey	J. P. Vansant, Dewey
	H. C. Weber, Bartlesville	E. E. Beechwood, Bartlesville
Washita	A. H. Bungardt, Cordell	James F. McMurry, Sentinel
Woods	D. B. Ensor, Hopeton	C. A. Traverse, Alva
Woodward	Joe L. Duer, Woodward	
	H. Walker, Buffalo	
	O. C. Newman, Shattuck	

Honorary Membership

In accordance with the provisions of the By-Laws of the Oklahoma State Medical Association, the following names of members in good standing of the Association, which have been placed on file in the office of the Association to be acted upon for honorary membership, are hereby published:

R. M. C. Hill, McLoud

Daniel F. Stough, Sr., Geary

S. P. Ross, Ada

J. C. Duncan, Forgan

Physicians Casualty Association Rates, Policies Unchanged by Emergency

"In these days when we are all confronted with a question of shortages in various commodities and an increase in the price of those obtainable, we are happy to announce that not only will we continue to carry our policyholders at no increase in the cost of their accident and health insurance but we adopted a resolution to the effect that there shall be no restriction under our policies by reason of Army, Navy or Marine Service and this is irrespective of where such Service may take the policyholder."—Physicians Casualty Association.

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Important to users of estrogens is the fact that Amniotin is now available in 10-cc. and 20-cc. diaphragm-capped vials. These new "bulk packages" provide two advantages . . . economy and convenience. The wide variation in requirements of women with menopausal symptoms can be met by simply withdrawing the proper dosage from the vial. The new vial packages provide a substantial saving over the cost of Amniotin in ampuls . . . without sacrifice of activity, uniformity or stability.

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Amniotin — A SQUIBB PREPARATION OF ESTROGENIC SUBSTANCES OBTAINED FROM THE URINE OF PREGNANT MARES

MEDICAL PREPAREDNESS

Procurement and Assignment Survey Being Made

The State Procurement and Assignment committee on March 5 mailed to the district chairmen questionnaires on all physicians of the counties in their respective districts.

In turn, the district chairmen are mailing the questionnaires to the Medical Preparedness chairmen of the county societies, asking that they immediately complete the request for the information contained therein, in order that the State Committee may have the necessary information on all physicians of the state when called upon by the Procurement and Assignment committee of the Federal government.

Every physician should bear in mind that this request is placing a tremendous responsibility on the County Medical Preparedness representative and that it is a responsibility that he did not ask for. The representatives were selected by the county societies themselves and with a full understanding that the representative and his committee might be called upon to act for the Office of Procurement and Assignment in evaluating the medical personnel of the county in relation to civilian medical care and military service.

World War II has made it necessary for an all-out effort on the part of every business and profession. Obviously, there will be instances where unanimous agreement cannot be obtained on either the method of procedure or the end result. Certainly no one can believe that any group or committee could better decide the needs of a county or community than those living and practicing there.

The procedure being followed is the democratic way. Give your support to the program and back the decision of your county society all the way.

Let us win this war.

State Committee on Procurement And Assignment Meets

The State Committee on Procurement and Assignment met February 8, at the Skirvin hotel, Oklahoma City, to discuss the work of the committee in the present emergency.

After hearing a discussion of the requests of the Office of Procurement and Assignment, as given by the chairman, Dr. Henry H. Turner, it was voted to utilize the chairmen of the Medical Preparedness committees in the counties of the state for the purpose of securing the necessary information on state doctors. The list of these chairmen appeared in the January issue of the Journal.

Each committee chairman has been sent a list of the doctors in his county and will be asked to determine the minimum number necessary for the protection of the health of the civilian population.

A similar survey was made in 1941, but cannot now be used as the basis of determining the medical personnel situation in the counties, as, since its completion, there have been changes brought about by death, removal and doctors being called into the armed forces.

The survey, when completed, will be utilized in advising the Office of Procurement and Assignment as to whether a doctor is available for military service. This method of procedure will obviously give the local doctors the opportunity of determining their local needs.

State Medical Preparedness Committee Changes Name

The Office of Procurement and Assignment, Washington, D. C., has requested of all State Medical associations that their committees on Medical Preparedness become the State Committees on Procurement and Assignment.

This request was immediately acted upon by Dr. Finis W. Ewing, President of the Oklahoma State Medical Association, and the Medical Preparedness committee will now be known as the State Committee on Procurement and Assignment.

The State Committee will continue to function through the County Medical Preparedness representatives, but the county committees will not be renamed.

One More State Doctor Called to Service

One more state doctor has been called to service by the Commanding General, Eighth Corps Area, according to the Journal of the American Medical Association.

Ordered to active duty is Dr. Lee Roy Wilhite of Perkins, who will be stationed as a Colonel at the U. S. Army Recruiting Station in Houston, Tex.

Three other state doctors whose orders to the service were revoked are:

Kinsinger, Ralph R., 1st Lieut., Blackwell

McCurdy, William C., Jr., 1st Lieut., Purcell

Preston, Thomas K., 1st Lieut., Muskogee.

Oklahoma Doctors Promoted in Navy

It has been reported to the office of the Association that Dr. Alton C. Abernethy, formerly of Altus, and Dr. M. P. Hoot, formerly of Ponca City, have been promoted to the rank of Lieutenant Commander in the naval service.

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No one physician, even if he were qualified, could afford to devote so much time and study to every new product. His Council renders this service for him, freely. Nowhere else in the world is there a group that performs the functions so ably served by the Council on Pharmacy and Chemistry and the Council on Foods.

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NEWS FROM THE COUNTY SOCIETIES

Dr. Richard L. Sutton, Jr., Kansas City, Mo., was guest speaker at the meeting of the Osage County Medical society, March 11, in Pawhuska. Before members of the society and guests from the Kay and Washington-Nowata County societies, Doctor Sutton discussed "Common Dermatoses."

About 12 members were present at the meeting of the Osage society, February 9, in Pawhuska. Guest speaker, Dr. Fred Glass of Tulsa, discussed "The Treatment of Burns" at this meeting.

"Diseases of the Rectum" was the topic of the talk which Dr. R. L. Murdoch, Oklahoma City, gave before the meeting of the Woodward County Medical society, March 12, in Woodward.

The society will meet April 9 in Shattuck, with Dr. Paul C. Colonna of Oklahoma City, and Dr. Paul H. Dube as speakers. Topic for discussion will be "The Diagnosis and Treatment of Fractures."

General discussion of "Congenital Dislocation of the Hip" and "The Selection of the Sulfonamide Drugs" followed the talk given on these topics by Dr. E. M. Gullatt of Ada at the meeting of the Pontotoc County Medical society, February 4 in Ada. About 13 members were in attendance.

Dr. E. A. Canada of Ada discussed "Meckel's Diverticulum" at the meeting of the Pontotoc society, February 18, in Ada.

Members of the Tulsa County Medical society are busy with arrangements for playing hosts to the Association's Annual Meeting in April.

The Tulsa county society met February 9 at the Mayo Hotel, with about 70 members in attendance. Major Louis H. Ritzhaupt, State Director of Selective Service, addressed the meeting on "Tulsa's Part in the Selective Service Program."

"The Use of Sulfonamide Drugs" was the subject of Dr. Charles M. O'Leary's discussion as guest speaker before the meeting of the Creek County Medical society, February 10, in Bristow. Doctor O'Leary discussed the treatment of peritonitis by the use of sulfanilamide and sulfathiazole in the peritoneal cavity, and the subcutaneous use of sodium sulfapyridine.

Dr. Frank Baum, McAlester, was presented with a silver plaque marking his fiftieth anniversary in the actual practice of medicine, at the Annual Inaugural Party of the Pittsburg County Medical society, January 28, in McAlester.

Dr. T. H. McCarley made the presentation of the plaque, and Dr. L. S. Willour presided as toast master for the party. Following the installation of new officers, Dr. Samuel Braden, pastor of the First Presbyterian church of McAlester, addressed the meeting.

A Symposium on Biliary Diseases was held at the meeting of the society, February 20, in McAlester. Doctor McCarley and Dr. C. E. Lively were in charge of this discussion.

Dr. Edward N. Smith of Oklahoma City was guest speaker for the meeting of the Cleveland county society, February 12, in Norman. Doctor Smith chose "Hemorrhage and Shock" as the subject for his talk.

About 20 members were present.

"Farm Security" was discussed by Dr. Charles M. Pearce before the meeting, March 11, of the Harmon county society, in Hollis.

Five members were present at the February meeting for an informal round table discussion of general problems.

A Symposium on "Lung Abscess," led by Dr. Walker Morledge and Dr. Charles M. O'Leary, was held at the meeting of the Oklahoma County Medical society, January 27, in Oklahoma City. About 75 members were in attendance.

Formal discussion of the topic was held by Dr. P. M. McNeill and Dr. J. H. Robinson.

Speakers for the program of the Oklahoma County Medical society meeting, February 24, in Oklahoma City, included Dr. L. J. Starry, Dr. N. Price Eley, Dr. L. C. McHenry and Dr. Henry H. Turner, all of Oklahoma City.

Doctor Starry discussed "Tumors of the Right Colon," and Doctor Eley led the discussion. Doctor McHenry discussed "Emergency Medical Service," and Doctor Turner gave a brief discussion of "Procurement and Assignment."

About 18 members attended the meeting of the Washington-Nowata county society, February 11, in Bartlesville, with Dr. S. A. Lang discussing "Allergy in Children" for the group.

The society is planning on presenting motion pictures, on the American Society of Clinical Pathologists and on Syphilis at a future meeting.

Dr. Henry H. Turner, Chairman of Procurement and Assignment, and R. H. Graham, Executive Secretary of the Association, both of Oklahoma City, were guest speakers at a meeting of the Seminole county society, January 14, in Seminole.

Doctor Turner discussed "Procurement of Medical Personnel for the Services," and Mr. Graham talked on "The Medical Profession's Part in Civilian Defense."

At the February 9 meeting of the Jefferson county society, a resolution was passed that the society instruct its delegate to the Annual Meeting to reduce the dues for the State Medical Association.

The society also passed a resolution that the Jefferson county society go on record as stating that participation in the County Health Unit will be discontinued as of July 1, 1942.

"Differential Diagnosis of Pulmonary Tuberculosis" was the title of the talk given by Dr. R. M. Alley of Shawnee, guest speaker for a meeting of the Garvin County Medical society, February 18, in Pauls Valley. About ten members were in attendance at the meeting.

Following Doctor Alley's talk, Dr. R. M. Anderson of Shawnee held an informal discussion. The Garvin county society will hold its March meeting at Pauls Valley, March 18, when a motion picture on Syphilis, sponsored by the Division of Veneral Diseases, U. S. Public Health Service, will be shown.

"Malpractice Insurance" was discussed by R. H. Graham, Executive Secretary of the Association, Oklahoma City, at the February meeting of the Kay County Medical society. The meeting was held February 19, in Tonkawa.

Dr. W. F. Bohlman outlined the work of the Committee on Emergency Medical and Hospital Care for members of the Blaine County Medical society at a meeting, February 19, in Watonga.

Members of the society will hear a refresher lecture on "Syphilis" sponsored by the State Health Department at their meeting March 19, in Watonga.

Plans are being made by the Pottawatomie County society for the Annual Spring Clinic. Serving on the committee in charge of preparations for this meeting are Dr. E. Eugene Rice, Dr. H. E. Hughes and Dr. Paul Gallaher.

The society met February 21, in Shawnee, with about 18 members attending the meeting. Dr. W. B. Mullins read a paper on "Recent Advances in the Treatment of Meningitis," and Dr. H. E. Hughes showed movies on "Appendicitis" and "Breast Tumors." The movies were shown through the courtesy of the Davis and Geck company.

Washington Birthday Clinic Attended by 77 Doctors

The Fourth Annual Washington's Birthday Clinic, sponsored by the Oklahoma City Internist Association, was held February 23, in Oklahoma City, with a record attendance of 77 doctors. Forty-five of the attending physicians were from over the state.

The success of the meeting was largely due to the organization work done by Dr. Mary V. Sheppard and Dr. Elmer Musick. Those appearing on the scientific program included Dr. P. M. McNeill, Dr. Bert F. Keltz, Dr. W. W. Rucks, Jr., Dr. Floyd Moorman, Dr. Wann Langston and Dr. William K. Ishmael. Those appearing on the Forum were Dr. A. W. White, Dr. L. J. Moorman, Dr. George A. LaMotte and Dr. Lea A. Riely.

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• OBITUARIES •

Dr. F. P. von Keller
1860-1942

Dr. Frederick P. von Keller, well-known pioneer Ardmore physician who was eminent in state medical circles for his outstanding work, died February 13 in the hospital which he founded and which bears his name.

Doctor von Keller was born, January 10, 1860, at Strasbourg, Alsace-Lorraine, France, of German-French parentage. Following his graduation from the the medical school of Heidelberg university in Germany, he came to the United States, where for a time he was medical examiner at Ward's Island, N. Y. Later he located in Galveston Tex., where he married Arkie Clay of Little Rock, Ark.

From Galveston, he moved to Chillicothe, Tex., in 1890, and four year later, he moved to Ardmore. Mrs. Arkie Clay von Keller passed away in 1936.

Doctor von Keller was a life member of the American Medical Association, a member of the Association of Military Surgeons of the United States, and served as president of the medical advisory board of Carter and Love counties, Oklahoma district No. 24, during the first World War. He was honored for his services in the United States Civil legion.

He was an associate professor of surgery in the College of Physicians, Dallas, from 1906 to 1907, and he was regarded as one of the best diagnosticians in the state.

He was also a member of the Beta class, the second consistory reunion held at the Scottish Rite consistory at McAlester, a member of the York Rite bodies, and a member of the Ardmore Masonic lodge No. 31.

In 1940, he married Mollie Dishman of Ardmore, who survives him. Other survivors include a daughter, Mrs. Donald M. Bretch of Oklahoma City; three granddaughters, Mrs. J. Samuel Binkley, New York City, Mrs. Donald Stuart Will, Champaign, Ill., and Mrs. Richard Gruner, St. Louis, Mo.; two great grandchildren, Donald James Frederick Binkley, New York City, and Gretchen Gruner, St. Louis; and two brothers, G. A. von Keller and L. V. von Keller, Greensburg, Kan.

Dr. John Frederick Kuhn
1872-1942

Dr. John Frederick Kuhn, another of Oklahoma City's Pioneer Doctors, has passed away. Time only will draw the curtain on the remaining few. A noble physician, a skillful surgeon, an honored gentleman and a faithful friend has died and thousands mourn his death.

Dr. Kuhn came to Oklahoma City in 1903, after finishing his residency at the Emergency hospital and at the Columbia Hospital for Women in Washington, D. C., where he trained under two great surgeons and gynecologists, Dr. J. Wesley Bovee and Dr. I. S. Stone. A few months after he established his office in Oklahoma City, where the Tradesman's National bank is now located, he married Miss Sadie Minker, of Norristown, Pennsylvania, who was a nurse he had met during his services at the Women's hosptal. Dr. and Mrs. Kuhn reared and educated their fine family in Oklahoma City. Dr. Kuhn saw our City grow to its present size. He was a loyal, progressive citizen, and possessed the qualities necessary for the expansion and culture of our beautiful City.

Dr. Kuhn was a busy doctor. His first many years, of course, were in the horse and buggy days. He was active in the promotion of good surgery in this locality and gave unselfishly his efforts to uphold the principles for its progress.

His ability was definitely recognized and no one ever questioned his loyalty and sincerity. His judgment was respected and his council secured in many of the difficult problems encountered by his colleagues. He was ready to help and give his assistance regardless of existing circumstances.

In 1912 he was appointed Instructor of Surgery in the Oklahoma School of Medicine and soon after became an Associate Professor in that Department. In 1925 he became Professor of Gynecology, which position he retained until a short time ago, when he was honored with the Emeritus Professorship. His department of Gynecology was his special pride and I believe it can be truthfully said that no one man's influence has meant more to the young doctors who have graduated from this school than that of Dr. John Frederick Kuhn's. His teaching and research activities and the countless hours given to this work were his contributions to the advancement of medical science. His desire to graduate good doctors was his daily ambition. He sacrificed the possibility of personal gain through the loyalty to the school and towards the progress of what he called the "boys."

In the passing of Dr. Kuhn, February 14, 1942, we lost a friend, a wonderful teacher, and one of Oklahoma City's greatest physicians. His devotion to his family was most beautiful. The hours spent with his students were innumerable. His interest in his private practice can be ascertained by his thousands of faithful friends and patients. His entire life was founded on the principles of being a "good man."—Joseph W. Kelso.

Psychiatric Institute Announced

Announcement has been made by the American Psychiatric Association of its forthcoming Fifth Postgraduate Institute for State Hospital men and other physicians interested in Psychiatry.

The Institute will open March 23 and will continue through April 3. It will be held at the Missouri State hospital, St. Joseph, Mo.

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* *Laryngoscope, Feb. 1935, Vol. XLV, No. 2, 149-154—Laryngoscope, Jan. 1937, Vol. XLVII, No. 1, 58-60 Proc. Soc. Exp. Biol. and Med., 1934, 32, 241—N. Y. State Journ. Med., Vol. 35, 6-1-35, No. 11, 590-592*

Auxiliary News

Haddon Hall will be the headquarters for the Annual Meeting of the Women's Auxiliary to the American Medical Association, which will be held at Atlantic City, New Jersey, June 8-12, 1942. Requests for reservations should be sent immediately to Haddon Hall, Atlantic City, New Jersey.

The Pittsburg County auxiliary met on February 3 at the home of Mrs. T. J. Baum, with Mrs. L. C. Kurykendall as co-hostess, for luncheon. Thirteen of the 14 members of this group were present. During the business session the following questions were discussed and action taken thereon: (1) $25.00 was sent to the Red Cross, (2) A $100.00 Defense Bond was purchased, (3) Twelve books were given to "Books for Victory." Three Red Cross Home Nursing classes are being taught. One class has been graduated. This auxiliary has also bought "Home Nursing Books" to lend to those persons who are unable to buy their own. The Pittsburg auxiliary is assisting with the feeding and clothing of a rural negro school.

On January 8 at 7:30 P.M. the Women's Auxiliary to the Cleveland County Medical Society met at the Central State Hospital, headed by Mrs. Jim L. Haddock, president. Eight of the 29 members were present. At this meeting the annual election of officers took place as follows: President, Mrs. Moorman Prosser; Vice-President, Mrs. W. B. Carroll; Secretary, Mrs. Curtis Berry; Treasurer, Mrs. W. H. Atkins. These members will take office as of September, 1942. Plans were also made at this meeting for a nurses' training course.

The Pottawatomie County auxiliary has a membership of 18 this year and meets in the homes of members. This group is headed by Mrs. Frank Keen, president. The Pottawatomie auxiliary has taken the following action during the year: $10.00 was given to Hygia; $12.00 was given to the Federal Nursing School and $10.00 was given for papers on "Tuberculosis Prevention" by seventh and eighth grade students. This auxiliary also has sent letters of thanks to Radio Stations WKY and KVOO in appreciation of the program "Doctors at Work."

Sulfadiazine Abstract Booklet A Convenience to Physicians

With the usefulness of Sulfadiazine growing rapidly, it was felt that a booklet containing abstracts of significant recent articles would be of real convenience to physicians and others working with this important Sulfa drug. Accordingly, Lederle Laboratories, Inc., New York, N.Y. have recently made available a 64-page booklet, "Abstracts Selected from Published Articles on Sulfadiazine."

In order to compress significant material into a booklet of this size, every article on the subject appearing up to December 1, 1941, has been examined. Articles which contained only minor evidence as well as those which overlapped or reworked prior authorities have been eliminated. At the same time, no attempt was made to select the most favorable articles. The result is a terse, but authoritative review of material on Sulfadiazine, arranged topically for convenient reference. In most instances selected articles have been condensed to one page or less. In every case, bibiliographies are supplied for those wishing to study the articles in full. This new booklet is available through Lederle representatives, or on request from Lederle Laboratories, Inc., 30 Rockefeller Plaza, New York, N.Y.

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